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Original Articles.

HEMISECTION OF THE SPINAL CORD.

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THAT the paths of voluntary motor impulse in the spinal cord are mainly, if not exclusively, direct, and those of sensation crossed, is the hypothesis which accords best with the facts of disease in man. There may be differences among the lower animals as regards the totality of the decussation of the motor tracts in the medulla oblongata, and of the sensory tracts in the cord itself, but the general law first laid down by Brown-Séquard has been fully confirmed by all the more recent and carefully conducted physiological experiments. While the paths of voluntary motor impulse have been satisfactorily defined by a combination of experimental, embryonic and pathological research, those of sensation have not been determined with the same degree of accuracy.

The most careful experiments on the lower animals, such as those of Ludwig and Woroschiloff, always leave room for doubt whether the reactions which result from the application of sensory stimuli to parts below the experimental lesion are to be regarded as signs of sensation proper, or merely of reflex action more or less general. On questions involving subjectivity the most satisfactory evidence would be furnished by observations in man, but we rarely if ever meet with spinal lesions so exactly limited as to be made available for this purpose. Of the numerous cases of unilateral spinal lesion on record, there is scarcely one of which, but for the aid of experi-

mental physiology, it could be said that the unilateral character of the lesion was at all certain. Most of them have not been investigated post-mortem, and of those that have been so, the investigation has been so imperfect, or the lesions have been so indefinite, that the unilateral character of the lesion has been assumed rather than proved.

I have only found one case in which the lesion was sufficiently definite and carefully investigated post-mortem; and even in this case the lesion was not strictly unilateral. The case in question has been described by W. Müller (Beiträge z. Pathologie und Physiologie des Rückenmarks, 1871). The patient was a woman who received a stab with a knife which traversed the spinal cord in a somewhat oblique direction below the third dorsal nerve, completely severing the left half of the cord, and also the right posterior column as far as the junction of the posterior root on that side. The division of the right posterior column therefore complicated a case otherwise definitely unilateral.

With the view of determining the effects of strictly unilateral division of the spinal cord in an animal as nearly human as possible I made the following experiment on a monkey.

The animal was chloroformed deeply, and, under stringent antiseptic surgical precautions, the cord was exposed in the region of the seventh and eighth dorsal segments.

Midway between the seventh and eighth nerves the left half of the cord was severed with a fine tenotomy knife. The incision was made, with the aid of a magnifying glass, from the posterior fissure outwards, without touching the right posterior median column.

The wound was carefully sewn up and dressed with antiseptic gauze and bandages.

The results described below were ascertained by daily observation and repetition of tests for a fortnight after the operation. During this time the general health of the animal was in the most satisfactory condition. On the fifteenth day the wound was examined, but it was found not to be united, though perfectly aseptic and free from discharge. It was again sewn up and dressed. Three days subsequently to this

the animal, which had continued apparently in good health, died suddenly.

On examination after death, an extensive clot was found pressing on the anterior surface of the pons and medulla oblongata. The hæmorrhage appeared to have originated from some of the lateral branches of the basilar artery.

The wound in the dorsal region was more or less gaping, but though there was some discharge in the dressings, the bottom of the wound and the spinal canal were perfectly free from pus. The pia-mater was somewhat thickened and adherent around the region incised, but the cord, with the exception of the lesion described below, was entirely free from injury or inflammatory softening.

The results of the lesion were as follows:—

On the side of lesion (left).

Mobility.—The left leg was absolutely paralysed as to motion, and not the slightest movement was made with this limb either independently or in association with the right leg. The leg was dragged when the animal moved about its cage with the aid of the hands and the right leg.

The temperature of the left leg was, during the first two days, much higher than that of the right. No exact thermometric determination was however made. When this was attempted on the fourth day, the difference, judged by the hand, was barely, if at all, perceptible, and the clinical thermometer, not graduated below 90° F., did not register on either side.

The patellar tendon reaction was at first greatly impaired or entirely abolished. While it was distinct on the right, none could be elicited on the left. Before death, however, the left patellar reaction was much more distinct than the right.

Sensibility.—The slightest touch or ruffling the hair of the left foot, flank, or left side of the tail, invariably attracted the animal's attention and caused it to look, put its hand to the part, or move out of its position as if wishing not to be disturbed. A prick with a pin, a pinch, or most of all, a touch with a heated wire caused it to rub the part, or angrily try to seize and bite the offending cause.

Repeated comparison of the reaction to painful stimuli of the hands with that of the left leg, did not however indicate any such difference as could be construed into the existence of hyperæsthesia of the left leg. If it existed, it was not shown objectively.

On the opposite side (right).

Mobility.—The right leg was freely moved volitionally in all directions, and for all purposes, without any appearance of uncertainty or ataxy. The animal could grip with its right foot with considerable vigour, and did so as well as with its hands, when e.g. it wished to free itself when held prisoner.

This however was true only when vision was free, and no impediment was offered to the intended movement of the right leg. When the animal was held prisoner and its eyes blindfolded, it was utterly unable to extricate its right leg from any opposition made to its intended movement, though as soon as the eyes were uncovered, it used the foot to grip and endeavour to free itself. The same inability to free the leg without the aid of vision was seen when the animal was crossing a ledge through a small door leading into another compartment of its cage. Happening to catch its toes on the ledge, it could only extricate the foot on looking round to the cause of the obstruction.

The manifest difference between the use of the right leg, with and without vision, clearly indicated the abolition of the sense of muscular contraction, and inability to appreciate the position assumed by the leg, except with the aid of vision.

Sensibility.—In addition to the abolition of the so-called muscular sense indicated in the preceding paragraph, there was total absence of any sign of perception of contact, ruffling the hair, pricking, pinching the toes or the belly of the muscles, or the application of a degree of heat sufficient to excite the most lively demonstrations of sensation in the hands or left leg.

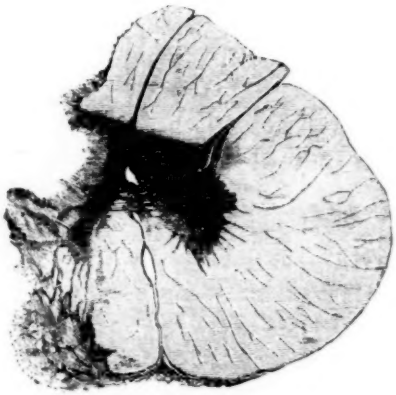
Not unfrequently, the momentary application of a heated wire to the toes or sole of the right foot was followed by a quick sharp movement of withdrawal. This was evidently purely reflex action, as the animal continued after this to allow the wire to remain in contact with the foot quite un-

concernedly, though its transference to the hands or the left foot at once elicited lively demonstrations of pain.

There was thus total anæsthesia and analgesia of the right leg; and this extended to the right flank and right side of the tail.

The power of evacuating the contents of the bladder and rectum was not perceptibly affected.

We have thus in this case complete motor paralysis, with retention of sensibility on the side of lesion, and complete anæsthesia and analgesia with retention of mobility on the side opposite the lesion.



The lesion.—The cord, otherwise intact, was found to be divided on the left side between the seventh and eighth dorsal nerves. The edges of the incision were slightly everted and sharply cut. No softening existed. The cord was hardened in bichromate solution before the exact extent of the incision was further investigated. In order to determine how far inwards it extended, the portion of cord including the lesion was immersed in a strong solution of logwood. This penetrated the lips of the incision, and tinged all the divided surfaces.

The transverse incision was then completed, and the exact extent of the primary incision accurately determined.

In the preceding figure (p. 5), which is a drawing by Mr. Collings, from a micro-photograph (magnified 12 diam.) of the first slice from the cord above the incision, the exact extent of the lesion is clearly displayed. The whole of the right side is intact. The posterior fissure is distorted to the right, so that the line joining the anterior and posterior fissures, instead of being straight, forms an arc of a circle with the convexity to the left. The left anterior column immediately bounding the anterior fissure—the anterior median column—is intact as far outwards as a line drawn from the anterior surface of the cord parallel to the anterior fissure through the apex of the anterior horn. A portion also of the anterior root zone external to this line was left undivided, but was almost severed from the anterior median column.

A wedge-shaped portion—the base anteriorly—of the left posterior column immediately bounding the posterior fissure was also undivided. The whole of the columns of the left side of the cord, with these exceptions, were severed. The blackened irregular-looking remnant, seen on the left side, is due to the logwood staining and irregularities of the divided surface. The grey matter round the central canal as well as the anterior commissure, and some portions of the grey matter of the anterior and posterior horns, were uninjured.

The internal boundary of the divided surface of the left half of the cord would correspond with a line drawn parallel to the anterior fissure, through the apex of the anterior horn, and gradually approaching the posterior fissure.

Microscopical investigation of sections of the spinal cord above and below the lesion, for which I am indebted to the assistance of my colleague Dr. Beevor, showed ascending degeneration, characterised by atrophy of the axis cylinders without any increase in connective tissue, in the left column of Burdach and in the direct cerebellar tract. For a couple of segments above the lesion an irregular wedge-shaped area, the base posteriorly, was visible in the column of Burdach less stained by carmine than the rest, owing to the almost entire absence of axis cylinders. In the cervical region the column was however entirely normal. No degeneration was visible in the column of Goll immediately above the lesion, or in the

cervical region, this portion of the posterior column having entirely escaped section or subsequent secondary lesion. There was extensive disappearance of axis cylinders, without sclerosis, in the left direct cerebellar tract above the lesion, and clearly visible also in the sections of the cervical region.

Below the lesion there was disappearance of axis cylinders in the postero-lateral column of the left side, corresponding to the area of the crossed pyramidal tract. No degeneration could be detected in the direct pyramidal tract, or in any of the other columns.

As in this case the right posterior column was absolutely intact, it is apparent that Schiff's doctrine that the posterior column is the path of tactile sensation on the same side, is erroneous. As the grey matter of the right side and also that surrounding the central canal on the left side was intact, it is also plain that these are not sufficient to convey the sense of pain on the right, for there was the most complete analgesia of the right leg. The experiment therefore is in absolute contradiction to the doctrines of Schiff, and entirely harmonises with those of Ludwig and Woroschiloff, which place the paths of voluntary motor impulse for the same side, and those of sensation for the opposite side in the lateral columns.

The motor path, the pyramidal tract, has been well defined; that of sensation, and its relations to the posterior horn and the tracts in its neighbourhood, require yet further elucidation.

If we compare the symptoms above described with those which are commonly said to result from unilateral lesion of the spinal cord in man, we find many points of agreement, but at the same time certain points of difference of an important nature and specially worthy of comment.

It is laid down by Brown-Séquard ('Lancet,' vol. ii. 1869, p. 869), who has been followed almost without question by most writers and text-books, that on the side of lesion there is paralysis of voluntary motion and of the blood-vessels, retention, in an exalted degree, of sensibility to touch, tickling, pain, heat and cold, but loss of the muscular sense; while on the opposite side there is conservation of voluntary motion and of the muscular sense, with total anæsthesia and analgesia otherwise.

As regards the total paralysis of voluntary motion and retention of sensibility on the same side, and retention of voluntary motion with anæsthesia and analgesia on the opposite side, the results of the above experiment are in complete harmony.

The fact of vaso-motor paralysis also on the side of lesion was confirmed by the evident increase of temperature in the left leg. This, however, was not persistent, in the same degree at least. Though the point was not investigated very minutely, there was (a few days after the operation) a very marked diminution of the difference in temperature between the two limbs which was at first so apparent. This would lend support to the views of Goltz and Vulpian, that the tone of the blood vessels is not entirely dependent on the vaso-motor centre of the medulla oblongata, but is capable of being maintained by the spinal centres themselves.

The existence of hyperæsthesia on the side of lesion was not evidenced by any noteworthy difference in the vivacity of response to sensory stimulation in the hand and foot respectively. But, on this point, the observations in man are so much superior to any inferences from the lower animals, and the existence of hyperæsthesia on the side of lesion has been so generally observed, that I should not regard the experimental facts above narrated as opposed to, even if they do not support, the existence of a condition of heightened sensibility.

As regards the muscular sense, however, the experiment is in diametric contradiction to the assertion that this sense is retained on the side opposite the lesion; and so far as I have been able to discover by examination of the published cases of hemispinal lesion, the assertion that the muscular sense is lost on the side of lesion, and retained on the opposite side, is not supported by any satisfactory evidence.

Most of what has been regarded as evidence of the muscular sense on the opposite side has no such meaning, and is of no value whatever in this relation. By the term muscular sense, is properly meant the knowledge we have of the degree of strain on our mobile parts, and of the position assumed by them actively, or given to them passively. It is usually tested by

the power of discriminating different weights or resistances, or by the capacity of judging blindfold the position in which a limb is during active or passive movements. It has however been regarded, by Brown-Séquard and others, as sufficient evidence of the muscular sense in the non-paralysed limb, that the individual is able to direct his movements aright. Thus of one of his cases he says, "the power of directing movements, which depends on the muscular sense, was markedly altered in the paralysed lower limb, while it was perfect in the non-paralysed limb."[1]

The power of directing movements, however, has no necessary connection with the muscular sense, and may be perfectly well retained when there is not a vestige of the muscular sense. During the acquisition of perfect volitional control over our limbs, the sensations arising in connection with muscular action no doubt largely help in guiding and directing our movements, but the aid of vision is also relied on to a much greater extent than is usually supposed.

The power of directing movements, once acquired, is capable of being exercised without any sense of muscular contraction whatever, and also without the aid of vision. Whether the intended movement has been actually carried out, however, the individual paralysed as to muscular sense is not aware unless his eyes are free.

In cerebral hemianæsthesia, even without the aid of vision, the patient is able to intend and carry out a given movement fairly well if no obstruction is offered. There is a remarkable case on record which proves that also, in total anæsthesia of spinal origin, the power of directing and duly co-ordinating the movements of the limbs is perfectly retained in the total absence of all sense of muscular contraction, and even with the eyes shut. In this case, recorded by Schüppel,[2] there was, in consequence of syringo-myelitis, total anæsthesia, and total abolition of muscular sense in the limbs, and yet the man could move his limbs vigorously, and carry out any desired movement with his eyes shut, without exhibiting any greater

[1] 'Lancet,' vol. ii., 1868, p. 594.
[2] 'Archiv d. Heilkunde,' vol. xv., 1874, p. 44.

degree of hesitation or caution than a perfectly normal individual would exhibit under similar conditions.

It is evident from these and similar facts, that the sense of muscular contraction is entirely separable from the motor innervation of the muscle. It is a complex of centripetal impressions generated by movements, active or passive, in the skin, joints, tendons, fasciæ and muscles themselves, in virtue of the sensory nerves with which these parts are furnished. That the whole of these nerves enter by the posterior roots and ascend in the cord entirely distinct from the paths of motor impulse has been proved anatomically, as well as by experimental and pathological evidence.

But, inasmuch as in cases of partial lesion of the sensory tracts there may be anæsthesia without analgesia, and analgesia without anæsthesia, it is theoretically possible that notwithstanding cutaneous anæsthesia, the sensibility of the muscles and deeper parts may continue, and thus form the basis of a sense of muscular contraction. But such a condition has never occurred in my experience, and I have nowhere been able to find satisfactory evidence to that effect. There is good reason for believing that, in all cases where the muscular sense has been retained, notwithstanding the abolition of the sense of pain and very great diminution of tactile sensibility, the power of localisation of cutaneous sensory impressions has remained acute. That this is possible has been well shown in a carefully investigated case recorded by J. Mackenzie,[1] in which, in spite of analgesia and anæsthesia to various forms of stimuli, the patient was able to localise with accuracy the slightest touch on the anæsthetic limb.

In a case reported by Brown-Séquard (Case 3, 'Lancet,' sup. cit.), in which it is stated that muscular sense remained notwithstanding the existence of almost complete anæsthesia and analgesia, it is expressly stated that the power of localisation continued perfectly accurate.

In Mackenzie's patient, when, at one period in the history of the case, the anæsthesia was absolute and the power of localisation abolished, the muscular sense was also entirely annihilated.

[1] A case of Hemiparaplegia Spinalis, 'Lancet,' June 9 and 16, 1883.

But, even if this power of localisation were inadequate to account for the existence of the so-called muscular sense in a limb otherwise anæsthetic, the retention of the muscular sense would, in view of the facts above related, indicate merely that the sensory tracts of the opposite side of the spinal cord had not been entirely interrupted, and would lend no countenance to the extraordinary doctrine enunciated by Brown-Séquard, that the paths of the muscular sense lie in the anterior roots and motor tracts of the spinal cord. This doctrine implies, and explicitly states, that the muscular sense is lost with the power of motion on the side of lesion. If by the muscular sense, we mean merely the power of directing movements, then, of course, this is lost on the paralysed side; but the loss is of motor power only, and not of the sense of movement. Naturally a patient cannot be made to estimate weights by volitional movements of a paralysed limb, but that he retains the muscular sense, so far as regards his knowledge of passive movements communicated to his limb, has been demonstrated beyond all doubt.

Few of those who have reported cases of unilateral disease of the spinal cord seem to have applied any proper tests on this point. But Köbner, in a case he has recorded,[1] found that in the paralysed limb, the perception of every movement communicated to it was perfectly retained, and the electro-muscular sensibility was in every respect normal. In Mackenzie's case also, where the limb was not entirely paralysed, the patient could appreciate weights quite correctly, so far as his feeble powers permitted; though he made mistakes when he attempted heavy weights, owing to the excessive sense of effort thus secondarily induced.

Brown-Séquard's dictum, therefore, that the muscular sense is lost on the side of lesion, is absolutely contradicted by facts such as these.

That the paths of muscular sense have any relation whatever to the anterior roots and motor tracts is opposed to every well-established fact of anatomy, physiology, and pathology of the spinal cord. Schüppel's case proves to demonstration that the paths of muscular sense, and sensibility in general, can be

[1] Deutsches Archiv f. Klin. Med., Bd. xix., 1877, p. 169.

entirely interrupted, while those of voluntary motion remain intact. The motor tracts and anterior roots in this case were perfectly normal.

That the muscular sense can be exercised, even to exact discrimination of differences in weight altogether independently of motor innervation, I have elsewhere shown.[1] In certain experiments made by Dr. Lauder Brunton and myself, it was found that the discrimination of weights when the muscles were made to contract artificially by the galvanic stimulus, was about as good as when the limb was moved volitionally.

As regards the mere knowledge of the position which a paralysed limb is made to assume by passive movements communicated to it, this, as Köbner's case shows, may be perfectly retained when the motor tracts of the cord are divided on one side. And it will doubtless be found, if the matter is investigated, that under such circumstances the patient will be able to discriminate differences in weight, by arrangements for exciting the muscles to action by electrical stimulation. But, indeed, so far as the knowledge of passive movements is concerned, the muscles, quâ their contractile elements, may be entirely eliminated without detriment. I have frequently demonstrated, in cases of long standing anterior poliomyelitis, in which all the muscular tissue of a limb had disappeared so that no contraction could be elicited by the strongest galvanic currents, that the patient so affected is able blindfolded to state with accuracy whether his limb is flexed, extended or otherwise moved. Under such conditions, as is well known, not only the muscular tissue, but the anterior roots and motor nerves are completely degenerated.

It seems unnecessary to multiply arguments further. Enough has, I think, been said to show the fallacy of Brown-Séquard's doctrines in reference to the paths of the muscular sense, and the way this is affected by hemisection of the cord. Hemisection of the cord paralyses muscular sense, as well as all other forms of sensibility, on the side opposite the lesion, and leaves it unimpaired on the same side.

[1] 'Functions of the Brain,' p. 228.

THE RELATIONS OF HYSTERIA WITH THE SCROFULOUS AND THE TUBERCULAR DIATHESIS.

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(*Continued from* Vol. VI. *p.* 460.)

II. Observations of Hysteria of a Tubercular Nature; with Pulmonary Manifestations: Reciprocal influence of the two Orders of Localisation.

Case XXVI. (condensed). (Obs. I. of the 'Iconographie de la Salpêtrière,' t. 1, p. 3.)—*Tubercular and Neuropathic Heredity. Hysteria, then Pulmonary Tuberculosis. Disappearance of the hysterical, coinciding with the apparition of the pulmonary phenomena. Death and Autopsy.*

L. Th., florist, 18 years of age. Admitted, August 1872. Father died at 42 years of age of a pleurisy; was very irritable and a drunkard; "nervous" attacks. Paternal grandfather neurotic; mother subject to bronchitis; maternal grandfather died in an apoplectic fit at 60 years of age; maternal grandmother died from phthisis, aged 46—had had twenty-one children.

During childhood, scrofulous accidents, bronchitis; of a teasing and malicious disposition. First hysterical attack in July 1871, from which time fits occur with all the typical characters: aura, hemianæsthesia, ovarian hyperæsthesia, etc.

Th. had always been chlorotic. From January 1876 had lost strength; vomiting and diarrhœa. In May, aggravation; rapid loss of flesh, with first symptoms of pulmonary phthisis, which carried her off on the 21st of July.

No hysterical attack occurred in 1876. In the beginning of May, hemianæsthesia and ovarian hyperæsthesia on the right side well marked. On June 1st hyperæsthesia less intense; pinching, pricking, cold, etc. perceived, but less acutely than on the left. Sight, smell, and taste, pretty equal on both sides, hearing less distinct on the right. July 13th: spontaneous hyperæsthesia has

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disappeared, ovarian pressure still painful, but no longer produced the sense of strangulation. General and special sensibility almost equal on both sides.

Autopsy: nervous system normal. Supernumerary pseudo-ovaries on the right. Adhesions at the apices of both lungs. Left lung: cavities in the upper third, granulations disseminated throughout the remainder. Right lung: several small cavities in the upper lobe; numerous tubercular masses elsewhere.

The authors of the 'Iconographie' add the following remarks: "An interesting feature of this case is the influence of tuberculosis, not upon the attacks—a well-known fact—but upon the permanent symptoms of hysteria, hemianæsthesia, and ovarian hyperæsthesia. We have seen how rapidly the latter symptoms disappear synchronously with the progress of the pulmonary lesions."

This is an important case for the view we are defending. There is a neuropathic and tubercular heredity. The diathesis is first localised in the nervous system, and is then manifested by the hysterical neurosis; next it invades the respiratory organs and determines a pulmonary tuberculosis. I look upon the hysteria and phthisis as mere successive manifestations of the hereditary diathesis, the symptoms of the one disappearing only upon the invasion of the other. An autopsy showed that there was true hysteria as well as true phthisis, and not tubercular disease of the nerve centres giving rise to hysteriform phenomena.

Such a case, observed in such a place as the Salpêtrière, will place in a better light our point of view than any theoretical discussion.

CASE XXVII. (condensed). ('Iconographie,' t. 11, p. 72.)—*Tubercular Heredity probable. Partial Epilepsy, Hysteria. Concomitant Pulmonary Phthisis, retrograding whilst the Neurosis persists.*

P. C. B., born in 1839. Admitted April 1862. Father a drunkard; died dropsical. Mother had hæmoptysis, died of bronchitis. Father and mother second cousins. Six brothers and sisters, all dead except one brother. Several attacks of bronchitis during childhood. First fit at 13 years of age; in 1860 had painful left hemiplegia. Coughs since four and a half years; had slight hæmoptysis twelve months ago. Frequent pains between

shoulders and under claviculæ. Expectoration abundant, mucopurulent, somewhat fœtid since three or four months. Occasionally marked dyspnœa, and fits of coughing, especially morning and night. Percussion is painful and shows dulness on the right, subclavicular in front, supraspinous behind. Humid rales at both apices, especially on inspiration, more marked on right side. Sonorous rhonchi elsewhere. Nocturnal sweats. In 1871 the state of the limbs varied. Left leg rigid; is the seat of occasional spontaneous trepidation, which is accompanied with tonic contraction of arm and rotation of head to the left. This is the incomplete seizure. In the complete attack the phenomena are more accentuated, and combine with rhythmical clonus. Flexion of the foot determines incomplete attacks. The patient sometimes passes whole days in a state of ecstasy, with occasional tremors of the limbs, but no general convulsion.

In 1872 there occur seizures in which loss of consciousness is not complete, the eyes closing upon the approach of the finger. On one occasion no fit, but peculiar state of abstraction; hallucinations of ear and eye.

In 1873 phenomena of the kind described as "hysteriform agitation" are described, reaching such an intensity as to necessitate recourse to the strait-waistcoat. After one of the attacks, patient is seized with laughter, stating that she hears what is said but does not understand it well; after another, during which the eyelids moved rigidly, a persistent contraction of the left leg ceases rapidly and sets in again an hour after. The patient also had convulsive attacks without cry or loss of consciousness, involuntary evacuation, nor foaming at the mouth, nor any consecutive stupor. Towards the end of the year distinct series of hysteriform fits.

From 1875 to, 1877 similar condition persists. Foolish and erotic delirium, then true "status hystericus." Hysterical and epileptic manifestations alternate with one another.

In June 1878 the author writes: "For a long time we have had no particular accidents to note with reference to the pulmonary organs. In the right apex, back and front, slight diminution of resonance, rough vesicular murmur, weaker than in the left. Coughs rarely." We need not give here a fuller account of the nervous symptoms, for which the reader is referred to the original.

This case is almost as remarkable as the preceding, although from a different point of view.

First it will be noticed that I place it under the heading of

Hysteria, although in the 'Iconography' it goes by the name of partial epilepsy. The details to which I have drawn special attention in the foregoing epitome, show that by the side of the partial epilepsy there really was a hysterical element in the patient. I should even be inclined to look upon the partial epilepsy as of hysterical origin, now that I have read the last note of Ballet and Crespin ('Progrès Médical,' Feb. 2nd, 1884) on the attacks of hysteria simulating partial epilepsy. But whatever view we take of it, the case can be utilised here.

It will be noticed that the tubercular heredity showed itself almost simultaneously in our patient by pulmonary symptoms and neurotic phenomena. Only it is a remarkable fact that instead of hysteria yielding to pulmonary tuberculosis, as was the case in the preceding observation, it is the phthisis which has given way to the hystero-epilepsy. For it is enough to compare the two examinations of the chest made in 1866 and in 1878, to see the enormous amelioration which has taken place in the phthisis which was already serious at the time of the first examination, and had been but very slightly treated in the interval. It is a pity that repeated examinations were not made during those twelve years, so as to show us the progress of the pulmonary lesions; but it does not prevent this observation from being of considerable interest.

CASE XXVIII. (Obs. IV. of Fouquet. Thesis; Paris, 1880, 343; p. 28.)—*Neuropathic and diathesic heredity (rheumatic and tubercular). Hysteria; stercoraceous vomiting, convulsions. Pulmonary tuberculisation—alternations between the nervous and thoracic phenomena.*

Heredity.—"Her father's death was caused by an accident. Her mother is very nervous. One of her sisters died phthisical at 18. Another is in pretty good health just now, but has had two attacks of acute articular rheumatism. This last sister is married, and has had two children who died of convulsions. She has also two brothers, both in good health."

History of the patient.—20 years of age; face yellowish, pale; characteristic aspect of chlorosis. Very nervous, laughs and cries without motives. Hysterical fit with stercoraceous vomiting. Very evident crepitations at the apex of the right lung. Later on, convulsive epilepsy with left ovaritis and hemianæsthesia.

"A fact worthy of notice is that when her attacks came back,

the cough was less troublesome and the state of the lungs seemed to improve; but as the patient was obliged to interrupt her work, she then took bromide of potassium; then, as the nervous accidents diminished in intensity, the state of the lungs grew worse again. In 1876 the two lungs were attacked, and in April 1877 the patient succumbed. She had not had any hysterical accidents during the last six months of her life."

Notwithstanding the absence of details, this summary brings into relief the same principles which were illustrated in Cases XXVI. and XXVII. We see here a very curious and repeated alternation between the hysterical and thoracic manifestations of the hereditary tubercular diathesis.

The author expresses himself clearly on the amelioration of the lung when the hysteria predominated. He shows the successive phases of the struggle, and the final victory of the pulmonary tuberculosis, which destroyed the patient after having altogether silenced the neurosis during the last six months of life.

Case XXIX. (condensed). (Obs. VII. of Briquet, loc. cit. p. 29.) —*Man; Neuropathic Heredity (father and sister), but doubtful as to the tuberculosis (mother). Hysteria; Tuberculosis. Death in Marasmus; tubercular granulations in apices of lungs.*

The mother, whose health was delicate, died in early life, of a disease which may have been either a nervous affection or a cancer of the stomach. The father died when 55 years of age, after two months of mental alienation with incipient paralysis of the lower limbs. His sister suffers from a nervous affection.

Always impressionable and easily moved; he has from 25 to 32 years of age been exposed to much worry. At that time various nervous phenomena appear. When 35 years old, convulsive attacks, leaving after them weakness and numbness in the left side of the body, then permanent pain in the middle of the back. He is admitted at "La Charité" when 37, in 1851.

The author describes the hysterical phenomena; then goes on as follows: "The thorax is about normal; resonance deficient under the right clavicle, and the vesicular sound is a little weak in that spot. From time to time there occur attacks of dyspnœa. No cough, no expectoration. When he exerts himself to cough, to swallow or to move about freely, one hears during inspiration a kind of stridor, resembling that of the first stage of a fit of

whooping-cough; suffocation seems imminent. At that time very frequent convulsive attacks, anæsthesia, etc.

"Gradually the nightly attacks become less frequent, but the patient got weaker and lost flesh notably. The difficulty experienced in the act of deglutition increased, the ingestion of food became more and more difficult. Gradually the respirations became embarrassed, the laryngeal sound increased, and at the same time the weakness rendered the cough, the expectoration, and the breathing more and more difficult. The continual epigastric pains and the nocturnal attacks have given rise to a feverish condition. Anæsthesia and paralysis have come back again more marked than before. The intellect, however, has always been wholly unimpaired. Death happened on May the 4th, after four months in hospital."

At the autopsy, nothing was found in the nervous centres except a little adhesion of the arachnoid and the pia-mater (thickened but transparent) to the brain.

"The lungs contain at the apices some miliary tubercular granulations." Nothing to note in other organs, except a slight thickening, of a lardaceous character, of the pylorus without noticeable stenosis."

We find again here, as in the three preceding observations, the coexistence in the same subject of hysterical and pulmonary manifestations. Here, too, the pulmonary tuberculosis progresses but slightly, and remains at the preliminary stage as in Case XXVII., the hysterical phenomena (at least the permanent phenomena) persisting, on the contrary, with great intensity.

This case differs from the preceding, however, inasmuch as the diathesis has killed the patient, whilst remaining but slightly marked in its thoracic localisations. The patient of Observation XXVI. died with the lesion of an advanced pulmonary phthisis; this one, on the contrary, with almost insignificant pulmonary mischief: he has succumbed to his general state, but not to the localisations of the diathesis.

Thus, in this patient, the constitutional affection has manifested itself both by the hysterical phenomena and by the thoracic symptoms. Owing to this double localisation, and to the predominance of the neurosis, the pulmonary manifestations have remained very slightly accentuated. But the progress of the diathesis has nevertheless continued, the

general nutrition has been altered, and the patient has succumbed in marasmus, thus plainly showing that the evolution of the tubercular diathesis is not necessarily parallel with the evolution of pulmonary tubercles.

CASE XXX. (condensed). (Obs. VI. of the Thesis of Chambard, p. 120.)—*Tubercular and Neuropathic Heredity. Pulmonary Tuberculosis; afterwards Hysteria.*

Father violent, alcoholic, and a libertine. Mother very nervous, dead of cancer of uterus. Many relations alcoholic. Brother dead of disease of the chest. Other brother, 20 years old, always ill, coughs much. Sister dead of disease of the chest.

Two years before her admission into the hospital (May 3rd, 1879), after a violent access of anger, pigmentation of face, which disappears rapidly with the application of tincture of iodine, but returns permanently four or five months after. Six months before, right pleurisy, which was rapidly cured.

"A month ago, following upon a chill, shivers and feverishness occurred, which kept the patient in bed four days; then she began to cough, and had two copious hæmoptyses. She also complains of profuse perspirations during the night, of vesperal feverish attacks and of loss of flesh; in a word, of the majority of the general signs of the pulmonary phthisis which the exploration of the lungs allows to detect easily." "It is because of these general disturbances brought on by that disease that she seeks admission to the hospital." "The expectoration is almost absent. The right apex shows evident signs of pulmonary tuberculosis. Harsh respiration, vocal resonance increased, rhonchus and lacunar crepitations. The same signs are present at the left apex, but less marked."

The report contains a detailed study of the hysterical phenomena observed or provoked in the patient. Unfortunately there is no information upon the ulterior evolution of the disease.

What is most interesting in this new example of coexistence of thoracic and hysterical signs in the same patient, is the antecedence of the pulmonary symptoms, whilst in the preceding case the neurosis occurred before the respiratory tuberculosis. Unfortunately this observation proves but little: first, because the hysteria is almost altogether a provoked one; and secondly, because no details are given concerning the ulterior history of the subject, and the respective evolution of the various symptoms.

CASE XXXI. (Largaud, Thesis of Montpellier, 1882, No. 23, p. 22.) —*Pulmonary Tuberculosis. Hydrotherapy. Hysteria. Disappearance of the pulmonary phenomena. Cure.*

"Miss X., age 20, was for two years under the care of Dr. Masson and Dr. Cavaillon for frequent hæmoptyses, with obstinate cough, intercostal pains, decline, and dyspeptic troubles. The auscultation revealed crepitations at the apices, which caused the doctors in attendance to diagnose incipient pulmonary phthisis. The ordinary treatment completely failed. Dr. Masson then ordered cold lotions to the extremities against the recurrent hæmoptysis, which ceased after a few days. The general state of the patient remaining always as bad, and the phthisis seeming to advance, Miss X. was submitted to a hydrotherapic course. After a shower-bath, a formidable hysterical fit broke out. A true neuropathic state manifested itself, and lasted a whole year, during which the morbid pulmonary phenomena improved in a marked way. A new affection then appeared which cut short the hysterical neurosis in the shape of a sciatica, which for eight months resisted every treatment. A furuncular eruption brought on a recovery from the sciatica, and the patient no longer showed anything special with reference either to the lungs or nervous system."

The succession of the symptoms here is very curious. Tubercular lesions in the lungs, then hysteria; the first symptoms disappear when the second begin. The sciatica takes the place of the hysteria, the boils take that of the sciatica, and lastly everything gets well. It is very difficult to look upon these as a series of distinct and antagonistic diseases, succeeding each other in the same subject. It is much more natural, and according to clinical experience, to look upon all these facts as being symptomatic manifestations, different and successive, of one morbid factor, the tubercular diathesis.

Let us notice finally the fact of the cold shower-bath bringing on the first attack of hysteria.

CASE XXXII. (Largaud, loc. cit. p. 30.)—*Pulmonary Tuberculosis. Hysteria. Succession of the two orders of manifestations.*

"During the year 1874, the woman B., 30 years of age, has spent 6 months at the hospital of Carpentras, with all the signs of a softening at the apices of the lungs (hæmoptysis, cough,

abundant nocturnal sweating, loss of flesh, dyspeptic troubles). She suffers afterwards of a series of most violent hysterical fits, and, during that time, the morbid pulmonary state improves from day to day. She leaves the hospital. Two years later, the woman B. has a child; she does not cough at all, and the most careful auscultation is negative. This woman has just had a fit of lumbo-abdominal neuralgia of indescribable violence coming after a chill, and simulating a myelitis of the lumbar region. She is pretty well to-day."

Given more in detail, this case (as well as the next) might prove interesting; epitomised as it is, we give it as simply indicating one fact to compare with the others brought together in this paper.

CASE XXXIII. (Largaud, loc. cit. p. 23.)—*Pulmonary Tuberculosis, then Hysteria.*

"The girl X., from 1871 till 1878, has spent three or four months of every winter in the hospital of Carpentras. She has had, first of all, many hæmoptyses, and presented sure signs of softening at the apices. The hysteria showed itself some time after (slight convulsions, globus, hiccough, etc.), since then, our patient, whose phthisis has improved, still comes back to the hospital every winter, showing signs of a general neuropathy, consisting in spasms, in a fidgety and talkative disposition, and in various visceralgias. She is always pale, thin, and has a characteristic dry and fitful cough. Auscultation, however, reveals nothing. She has a special appearance (vague look, dilated pupils, and tendency to ecstatic attitude). The girl X. is still alive to-day."

CASE XXXIV. (Largaud, loc. cit. p. 24.)—*Pulmonary Tuberculosis, then Hysteria.*

"The girl M. has at first presented all the symptoms of a pulmonary tuberculosis progressing slowly (cough, hæmoptyses, considerable loss of flesh, nocturnal sweating). Seized with a violent hysterical fit, followed by many others, the phthisis improved and disappeared, giving place to a hysterical paraplegia, with prolapsus recti, which for five years resisted all treatment. The cure of her paraplegia has made a great sensation, for it is, she says, after nine days' prayers at 'Notre Dame de Santé,' that she has been cured. In the month of July 1880, she wanted an operation to be performed for the prolapsus recti. After resection,

peritonitis set in, and she died four days after. The autopsy could not be made."

CASE XXXV. (Largaud, loc. cit. p. 25.)—*Pulmonary Phthisis and Hysteria. Alternating predominance of the two orders of symptoms. Tubercular Cachexia; Diarrhœa; Death.*

Philomène S. remained one year in the hospital of Carpentras, for nervous attacks.

"Patient is a foundling; she is twenty; during the time of her youth she has been pale, puny, and has presented sure signs of scrofula (ganglionary swellings of neck, very obstinate eruption on the head, etc.).

"During her one year's stay at the hospital, she has presented alternations of obstinate cough (we cannot here define the state of the lungs) and hysterical fits. These were either powerful attacks with globus and convulsive movements, or mere adumbrations of fits. The whole course was very irregular. She left the hospital very much better, as far as the cough and the neurosis were concerned, but her general state was still very bad.

"Philomène married some time after, and gave birth to a child which only lived a few days.

"She was lost sight of up to the day when she was admitted into the hospital for an obstinate diarrhœa (January 3rd, 1880).

"She says that since her confinement she has had a few fits, which have diminished in number and in intensity from the time when the cough has come back stronger, and the diarrhœa manifested itself.

"She is very much thinner, very pale, and her courses have stopped. The auscultation reveals crepitation at both apices, especially on the right side, and prolonged expiration. At night, when the respite given her by the cough allows her to sleep a little, she is at once bathed in perspiration. She has as many as ten motions a day, especially in the morning, and the fæces present the appearance of clay, and are of the consistence of pap. They are almost odourless, and contain neither shreds of false membranes nor any traces of blood. As to the neurosis, its only manifestation is a certain mental alternative. She chatters incessantly, tells lies at every opportunity, and passes without reason from laughter into sobbing fits. Not the slightest appearance of convulsive attacks.

"The hysteria remains hardly noticeable for three months, and would perhaps have been overlooked, but for the previous history; but auscultation and expectoration give us daily fresh evidence of

the progress of the phthisis, so much so, that from day to day a fatal termination is apprehended.

"On April 10th, we are called at midnight. The following is the state in which we find her: She is shaken by movements so inordinate that all the nurses can do is to keep her in bed. She cannot speak, but she puts one of her hands to her throat, and seems to beg that the assistants would tear away something which grasps and chokes her. The eyes are haggard, respirations noisy and stertorous. Then begin clonic convulsions which last for about a quarter of an hour, after which the patient, exhausted, falls to sleep till the morning.

"There we have evidently a return of the hysteria with a regular attack. From that day she has nothing but fits declining in intensity (8 to 10 in number), and we notice with astonishment that all the phthisical symptoms are daily improving. On the 1st of September, the lungs have nearly recovered their natural action, and the patient has regained a little stoutness and strength; although the diarrhœa persists (3 to 6 motions in 24 hours), resisting all the treatments directed against it.

"In the month of March of the following year, Philomène leaves the hospital, still with her diarrhœa, and goes back to her occupations.

"We have since heard that the patient, again considerably weakened by the diarrhœa, has once more been admitted into the hospital during the first days of January 1882. Her hysterical attacks had completely ceased, and the diarrhœa had given place to a generalised œdema to which she succumbed on the 1st of March, without other characteristic symptoms of tuberculosis.

"We regret that the autopsy could not be made."

What strikes one in this case, as Dr. Largaud says, "is the slowness with which the symptoms of phthisis have progressed in a patient so evidently affected by the tubercular diathesis.

"At first both phthisis and neurosis progressed together, a kind of equilibrium being produced between the two affections. Later on, the pulmonary phthisis manifested itself in a formidable outburst. It then stopped almost suddenly, and at the same time the hysterical attacks of the greatest intensity began.

"The thoracic accidents then gave place to an intractable diarrhœa, evidently tubercular in character, and which in the end carried off the patient."

CASE XXXVI. (Largaud, loc. cit. p. 28.)—*Pulmonary Phthisis at the beginning; Hysteria displacing the thoracic symptoms.*

Marie X., age 27, admitted into the hospital of Carpentras the 3rd of February, 1881. Cannot give any information concerning her heredity. Seems to have had syphilis when 21 years old; but no trace of it is found at the time of her admission except a chronic laryngitis with very harsh voice. Rheumatic pains (?) for last two months. She has taken a violent cold, degenerating "into an acute bronchitis, which assumes from day to day more the tubercular character; first sanguineous expectoration, then veritable hæmoptysis; abundant nocturnal sweatings; noticeable weakening and loss of flesh. The auscultation reveals dry and very harsh crepitations at both apices, and prolonged expiration. Percussion yields a great difference of resonance between the apices and the bases, especially on the right side. Fever."

"Patient was growing daily worse, when about middle of April she was suddenly taken, at two o'clock, P.M., with a violent hysterical fit. She was near the stove when she felt herself getting weaker, to such an extent that she had to be carried to her bed. She then experienced in the epigastric region an abnormal weight, as if something were rising, clutching her at the throat and choking her. She is extremely agitated, screaming aloud, then falling as if she were lifeless. Convulsions supervened, consisting of pretty regular alternations of contraction and of flexion of the legs and arms, then movements of pelvis ('cynical phasis'). After a few moments, the patient, exhausted, falls asleep.

"The next day, a second fit at the same time; then a third and a fourth one, all identical. Since that time the state of our patient has altogether altered. The pulmonary affection improves very rapidly, so much so, that after a month every trace of phthisis seems to have disappeared."

"The hysteria goes on, but is of a benignant type (mind, neuralgia, anæsthesia, etc.), and the patient leaves the hospital in September, keeping nothing else of her neurosis than certain mental peculiarities."

This observation has not perhaps the importance given it by Dr. Largaud, because the pulmonary phthisis is insufficiently proved. Nevertheless we see that instructive succession of phenomena: thoracic troubles exhibiting the signs of an in-

cipient pulmonary phthisis, the place of which is afterwards completely taken by a marked hysteria which sets in suddenly.

CASE XXXVII. (Largaud, loc. cit. p. 81.)—*Incipient Pulmonary Tuberculosis, replaced afterwards by Hysteria.*

Louise B., 17 years old, "assistée" child of the Hospice, is admitted into the hospital of Carpentras on March 4th, 1881, with all the symptoms of chloroanæmia. Douches and ferruginous preparations. After sixteen days of this treatment, she is taken with violent cough and sanguineous expectoration; at the time of admission auscultation had revealed nothing. Notwithstanding the suppression of treatment the patient coughs more and more, and crepitations are heard in the whole apex of the right lung, as well as harshness of the respiratory sounds, and prolonged expiration on both sides. Hæmoptyses, nocturnal sweatings. The patient, very much weakened, can no longer leave her bed. The diagnosis made is tubercular bronchitis.

Things grow worse till the 1st of May, at which time the patient is suddenly taken, during the morning visit, with slight convulsions, followed by deep prostration. On the same day, at 3 and 10 in the evening, other fits like the first.

The next morning at the time of the visit, aphonia, which persists with alternations. Slight convulsive fits every day, and constriction of the throat, anæsthesic and hyperæsthesic regions. This condition persists for two weeks, and at the same time there is a marked improvement in the respiratory system, and the general state. Then about the middle of June, the hysterical phenomena improve in their turn, and on July the 1st, Louise B. seems quite cured, and asks to leave the hospital. She goes out on the 1st of September, at which time the symptoms of pulmonary phthisis have disappeared, and of the neurosis there only remains a great tendency to lying and chattering.

The author sees the patient again in March 1882, and finds nothing in the lungs, but during the last month she shows fresh symptoms of the neurosis. She suffers every other day from formidable hysterical fits, with globus, tonic and clonic convulsions, erotic in character, and with loss of consciousness. The compression of one of the ovaries calms the fit almost instantly, whilst the compression of the other increases its intensity, and draws loud screams from the patient.

With the same reservations that we have made concerning the preceding observation, we have here again a remarkable

example of thoracic phenomena, probably tubercular, replaced later on by hysterical manifestations. Let us add that the rapid disappearance of the latter was only an apparent recovery, and that the hysteria had really taken possession of the patient with its usual characters of permanence and tenacity.

CASE XXXVIII. (Largaud, loc. cit. p. 36.)—*Tubercular Heredity; Pulmonary Tuberculosis; disappearance of those phenomena and appearance of fits of Somnambulism.*

Rose C., 7 years of age. Father and mother dead in 1879 of pulmonary tuberculosis. She, after a cold, becomes feverish, has an obstinate cough, slight hæmoptyses, nocturnal sweatings; loses flesh exceedingly rapidly. At first crepitations, then evident signs of softening in the right apex. Three times these symptoms abate, and then reappear. At last, eight months after the beginning, marked and lasting amelioration. There only remained, to the great astonishment of the doctors, slight neuralgia in the intercostal spaces and in the legs.

"One evening the sister calls us to show us Rose in a fit of somnambulism, walking in the ward with her eyes shut, and stopping at every bed to fill the patient's glasses with medicine; she goes by us without seeing us, and goes back to bed without saying a word. The sister informs us that similar somnambulistic attacks had already been noticed of late." Rose leaves the hospital in November 1880, in a very satisfactory state.

Here the pulmonary tuberculosis is hereditary, it begins with unusual intensity for a child 7 years old. Afterwards she recovers against all hopes, and fits of somnambulism appear.

"We think," Dr. Largaud says, "that the cure of the phthisis in this child of 7, phthisis which seemed likely to go through its different stages in a very limited time, is much less due to the treatment followed, than to the appearance of a neurosis, which at first has checked and afterwards removed the thoracic accidents."

We reserve the theory of Dr. Largaud for further consideration, but the fact itself is very important as far as our argument is concerned.

CASE XXXIX. (condensed). (Berdinel, 'Arch. de Médec.' 1875. Chataing, Thesis, Paris, 1880, No. 213.)—*Heredity unknown. Hysteria. Beginning of Pulmonary Tuberculosis which does not progress.* (*The observation includes a space of* 8 *years.*)

Lecomte (Marie Elisabeth), 29 years of age. Admitted into the hospital Cochin, the 29th of May, 1873.

Foundling. No previous history, except a typhoid fever in 1870 or thereabout. In April 1873, right pneumonia. Persistent anuria after the application of blisters. In June, symptoms of pelvi-peritonitis. The general state is disturbed, and at the same time the patient becomes whimsical, etc. The vague nervous troubles accentuate themselves. Pains in abdomen; vomiting. In November, sleep of three days. The nervous phenomena become more marked. The anuria persists.

In January 1874, left hemianæsthesia. Anuria and vomitings. Recurrence of pelvi-peritonitis. In June paralysis of left leg. On the 16th of August, beginning of pleurisy on the left side with effusion. In October, anæsthesia and paralysis become paraplegic. Sometimes in the beginning of December, a few fits of coughing, followed by slight hæmoptyses accompanied by thoracic pains. However, anuria and paraplegia persist.

1*st January*, 1875.—Patient has a tired look, is thin; she has coughed a little for some time back, and has had some hæmoptyses; auscultation reveals at the right apex a few dry crepitations; still paraplegia with anæsthesia. Voice harsh, almost toneless for more than three months. Vomiting and ischuria. The hysterical state shows itself also by a marked mental peculiarity, by diffuse neuralgia, by feelings of suffocation which, for some weeks past, have taken the characters of the usual globus; profound anæsthesia of the ocular and pharyngeal mucous membranes. Semicataleptic sleep in March. Later on, pulmonary congestion of the left side, with fever. In April, long fits of sleep for 17 days. (See the very carefully analysed details in the work of M. Berdinel.) The patient leaves the hospital on July 29th, and M. Berdinel adds: We have since seen the patient; she does her work, which however is not trying, fairly well, but she shows signs of pulmonary tuberculosis. Her voice is rather hoarse, and she vomits from time to time after food.

The patient does well for four years, except for an attack of erysipelas of the face. On March 10th, 1880, she is taken with pains in the throat and with aphonia, then the urinary troubles reappear; vomitings, bilateral ovaritis, right hemianæsthesia.

Sensory disturbances. In the midst of the history of the progress of those nervous phenomena we find (April 28): The patient looks well, but she always perspires very freely. She complains of a slight cough, but auscultation reveals nothing but a little respiratory harshness at the apices. On May the 13th, she leaves the hospital, suffering only from aphonia.

In that celebrated hysterical patient, whose fits of sleep at the hospital Cochin have created a great sensation (see, besides the paper of Berdinel quoted previously, that of Monod in the same journal, 1877), the thoracic and neurotic manifestations exist together and complicate one another. Alternation is therefore not observed in her case, the two orders of symptoms do not take each other's place. Only the tubercular lesions, instead of going forward and progressing, remain stationary, and even retrograde during that long period of eight years, during which she is closely observed, and during which the various manifestations of the neurosis predominate greatly.

This observation differs from the majority of the preceding in certain particulars; but it is connected with them in other aspects, and establishes at any rate a clinical type worth remembering.

(To be concluded.)

ON THE CONDITION OF THE FUNDUS OCULI IN INSANE INDIVIDUALS.

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SINCE the introduction of the ophthalmoscope, the investigation of the condition of the fundus oculi in insane individuals has occupied the attention of many observers; though in this country, at least, the published observations are not very numerous. It cannot be said, however, that the subject is one which is yet by any means fully elucidated; indeed, the discrepancies between the recorded observations of different investigators is so great, that to reconcile the opposing statements is no easy task. With the view of endeavouring to throw some light on the question, the investigation shortly to be described was undertaken, and though we are sensible that the number of observations is too small to admit of anything like a settlement of the points in dispute, we think that a record of them will not be without its value.

It need hardly be said that in many insane individuals, the use of the ophthalmoscope is attended with much difficulty; some patients give trouble by their restlessness and turbulence and by the impossibility of fixing their attention; others again become much alarmed when they are brought to the "dark room," and no persuasion or assurance will get them to submit to an examination. By taking care however, that the pupils are thoroughly dilated with atropine, and by the use of patience and perseverance, these difficulties can in the great majority of cases be overcome. We have, however, in a few instances, been compelled to abandon the examination as altogether impracticable, and in a few other cases, only an imperfect view being obtained, we could not feel certain

of the result; all such cases are however excluded from the descriptions which follow, which deal only with those about which we feel that we can write in full confidence.

Our observations have been conducted with the most recent refracting ophthalmoscopes, the "indirect" method having been in almost every case supplemented by the "direct;" we have also, in the majority of cases, taken care to note such errors of refraction as existed, and have endeavoured to measure the amount of these as closely as possible. In the case of General Paralysis we have further supplemented the investigation by the use, wherever practicable, of test types, with which we have obtained reliable results in a much larger proportion of cases than we anticipated when we commenced. It may indeed be objected by some, that subjective examination in persons mentally alienated can never give results of scientific value, but this objection applies only to the negative aspect of the question; if a lunatic *reads* correctly certain test types it is obvious that he can *see* them, whereas the failure to read them wholly or in part, does not necessarily imply inability to *see* them, since his incapacity in this respect may be due to lack of attention, defect of mental power, or other cause. We should have thought indeed that the careful use of test types as a supplementary aid in an investigation of the kind before us scarcely required defence, were it not that no less an authority than Dr. Clifford Allbutt has stated[1] "that failure of sight as tested by types is wholly an oculist's symptom, and bears little proportion to the amount of atrophic or other disease that may be seen in the nerve." It is indeed well known that a patient's sight may be perfectly intact, even when intense papillitis is disclosed by the ophthalmoscope, the explanation being that the conductivity of the nerve tubules is as yet unimpaired; and we quite admit that it is often impossible, from a mere inspection of an atrophied disc, to pronounce definitely as to a patient's acuity of vision, but we think that the cases of optic nerve atrophy have yet to be described in which a patient is capable of reading J 1 and $\frac{20}{20}$, and in a doubtful case where the diagnosis lies between a slightly atrophied and an anæmic optic disc, we think the use of test types may often give valuable help. And this leads us to

[1] 'The Ophthalmoscope,' p. 380.

make a few remarks on the normal colour of the disc; it can, we think, hardly be doubted, that the earlier observers took too rigid a view as to the physiological differences in colour here displayed; it was not fully recognised, and is even now, we think hardly sufficiently so, that the colour of the disc may vary in different individuals from an almost uniform redness to an almost uniform pallor, and yet the disc in question may be in a perfectly normal condition. We have an instructive case under observation now, in the person of a female epileptic whose discs are of an almost uniform whiteness strongly suggestive of atrophy, and yet the patient can read *perfectly* J 1 and $\frac{20}{20}$. In fact we cordially endorse the statement made by Dr. Gowers,[1] "that the tint of the optic discs may vary as much as the tint of the cheek."

We wish it had been possible to have in addition tested the field of vision, but we do not consider that reliable results could be obtained by this method in the persons of lunatics.

In some of the cases of General Paralysis, we have made a microscopical examination of the optic nerves and discs after death, but we have not thought it necessary to insert any account of the naked eye appearances of these parts, as we consider that these appearances furnish *usually* but a very rough guide to the intimate structure, and are therefore not to be relied upon.

Our observations have all been made in the wards of Rainhill Asylum, and we are indebted to Dr. Rogers, Medical Superintendent of that Institution, for the facilities afforded us in the examination of patients. The investigation has extended over a period of two and a-half years, and has embraced cases exhibiting every variety of mental disorder. The results will be given in detail as we proceed, but we may say at once that the actual lesions noted have been comparatively few.

A word or two first, however, with respect to the classification of mental disorders here adopted. For a purpose such as that before us, we think that the simpler we can make this, the better, and the old division of Insanity into Mania, Melancholia and Dementia, has at least the advantage of simplicity, and the further one of being in possession of the field: many varieties of mental disorder are, however, included under each of these heads which cannot in strictness be grouped together,

[1] 'Medical Ophthalmoscopy,' 2nd edition, p. 4.

and therefore, whilst retaining it as our basis we have elaborated it in detail. In the first place then, we divide all our cases into General Paralysis, and Not-General Paralysis; and the latter class we subdivide as follows: of Mania we make four subdivisions; "Acute" including all recent cases slight and severe; "Puerperal," which is of course simply a variety of acute, but to which it is perhaps better to assign a separate heading; "Chronic"; and the "delusional form," which includes the cases commonly known as "Monomania." Of Melancholia we make a threefold division, into "active recent," "passive recent," and "passive chronic."

Cases of Mental Stupor we include in a class by themselves. Cases of Dementia are subdivided according as they are "Primary" (including what is known as Acute Dementia whether recent or chronic), or secondary; the latter again into cases secondary to Mania and those due to gross organic disease of the brain, such as old Cysts, Hæmorrhages &c., and which may be styled "organic;" whilst a fourth subdivision comprises all "senile" cases, *i.e.* cases of primary dementia in old people not known to be due to gross changes.

We make a separate division of Epilepsy, which again we subdivide according as it is associated with imbecility—with slight—or with grave—impairment of the mental faculties. Cases of Imbecility, uncomplicated with Epilepsy, are also included under a separate heading.

Our classification will then stand thus:—

I.—General Paralysis.

II.—Not-General Paralysis
- 1. Mania
  - *a.* Acute.
  - *b.* Puerperal.
  - *c.* Chronic.
  - *d.* Delusional form of.
- 2. Melancholia
  - *a.* Active, recent.
  - *b.* Passive, recent.
  - *c.* Passive, chronic.
- 3. Mental Stupor.
- 4. Dementia
  - *a.* Primary (recent and chronic)
  - *b.* Secondary to mania.
  - *c.* Organic.
  - *d.* Senile.
- 5. Epilepsy
  - *a.* With imbecility.
  - *b.* With slight dementia.
  - *c.* With grave dementia.
- 6. Imbecility (uncomplicated with epilepsy).

Taking all forms of Insanity together, we have examined 313 different cases (persons); of these 66 were General Paralytics, and 247 not-Paralytics; of these latter in 204 (82·59 per cent.) the optic discs, and main retinal vessels were perfectly normal, whilst in the remaining 43 (17·40 per cent.) these parts were either abnormal or were marked as doubtful, *i.e.*, in these latter the changes were so slight, that no decided opinion as to their abnormality could be formed. In some of the abnormal cases again, the changes noted (as will be seen in detail as we proceed) were clearly due to causes such as Bright's disease, which had no connection with the patient's lunacy or at best but an indirect one; and these cases must of course be allowed for, in endeavouring to trace a connection between the patient's mental (cerebral) state, and the condition of his optic discs or retinal circulation. Furthermore, as is well known, a variety of functional constitutional states are at times associated with changes in the discs, altogether apart from the question of lunacy. The effects of errors of refraction have also not unfrequently to be discounted. If due regard be paid to these considerations it will be found that the percentage of cases in which we have noted a distinctly abnormal condition of the discs, &c., in connection with mental alienation (other than that associated with General Paralysis) is a very small one.

We will now proceed to give in detail the observations we have made under each head; after which we purpose briefly to refer to the more important communications on the subject that have appeared in this country, and finally to draw some conclusions.

We will begin with cases of General Paralysis, this affection being not only the most important, but that in which we found by far the largest proportion of departures from the normal condition.

[Editorial Note.—Circumstances independent of our own will have compelled us to let the main portion of this paper, and the Plates which illustrate it, stand over till our next issue. The following Tables contain a synopsis of the cases referred to by the Authors.]

TABLE I.—Showing the Condition of the Eyes in

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
1	E. McM.	F.	24	1st	'81, Oct. 29	Blurring of inner edge of each disc (most marked in left).
				2nd	'82, Mar. 22	Same condition.
				„	„ Nov. 4	„ „
				„	'84, Feb. 2	Condition absolutely unchanged.
2	H. B.	F.	44	1st	'81, Nov. 7	Both discs normal; arteries very small, and few in number.
				„	'82, Mar. 15	Both discs pale.
				„	„ „ 29	„ „
				2nd	„ June 10	„ „
				3rd	'83, Jan. 25	„ „
3	A. B.	F.	56	2nd	'81, Nov. 2	Both discs perfectly normal.
				„	'82, Mar. 15	„ „ „ (pink).
				„	„ Sept. 14	Both discs very red, nearly the colour of the fundus; redness invades centres; margins of left disc decidedly blurred.
				„	„ Nov. 23	Same condition.
4	A. R.	F.	38	1st	'81, Nov. 2	Both discs perfectly normal (red). Large staphylomata around each, developed chiefly to inner sides.
				„	'82, Mar. 15	Same condition.
				„	„ „ 29	„ „
				2nd	'84, Feb. 2	„ „
5	A. W.	M.	42	2nd	'81, Nov. 5	Both discs perfectly normal.*
6	E. L.	F.	33	2nd	'81, Oct. 16	Both discs perfectly normal. (Indirect examination only.)
7	T. C.	M.	41	2nd	'81, Nov. 9	Both discs perfectly normal.
				„	'82, Mar. 11	Same condition.
8	J. O.	M.	37	2nd	'81, Nov. 10	Both discs perfectly normal. (Peripheral striæ in both lenses.)
				„	'82, Mar. 11	Same condition.
9	T. P.	M.	32	2nd	'81, Nov. 10	Right disc perfectly normal. (Left eye—opaque capsule.)
				„	'82, Mar. 11	Right disc perfectly normal.
				„	'84, Feb. 20	„ „ „
10	J. C.	M.	43	2nd	'81, Nov. 10	Both discs perfectly normal. Numerous small pigment spots in right retina.
				„	'82, Mar. 11	Same condition.
11	F. W. T.	M.	43	2nd	'81, Dec. 17	Both discs perfectly normal. A small patch of choroidal atrophy in vicinity of right macula.
					'83, Jan. 5	Same condition.
12	E. D.	F.	21	2nd	'81, Dec. 7	Both discs perfectly normal. (No staphylomata.)
13	C. B.	F.	42	1st	'81, Dec. 3	Both discs decidedly red, the area of greatest redness being a middle zone between centre and margins, which are pale.
				„	'82, Feb. 1	Both discs very red, almost the colour of the

* In this and all other cases where no special reference is made to the condition

SIXTY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE.

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
R. + 3 D. L. + 2 D.	R. $\frac{20}{15}$ J. 1 L. $\frac{20}{30}$ J. 1	3½ mm. equal, regular; act well to light and to accommodation.	Still under observation.	The blurring of inner edge of each disc is probably a physiological condition.
Emmetropic	..	R. 5 mm., L. 3. regular; very little action either to light or to accommodation.	Died, '83, July 26.	Had aortic regurgitation.
R. + 1 D. L. + 1 D.	R. $\frac{20}{15}$ J. 16 L. $\frac{20}{30}$ J. 16	R. 3 mm., L. 4 mm., both regular; sluggish to light, but act to accommodation.	Died, '83, Jan. 20.	Died of phthisis. Microscopical examination of optic nerves, &c. Pachymeningitis of cerebral dura mater.
R. − 10 D. L. − 10 D.	R. $\frac{20}{6}$ J. 2 L. $\frac{20}{6}$ J. 2	R. 3 mm., L. 4 mm., regular; act to light and to accommodation.	Still under observation.	
..	..	.. ..	Died, '81, Nov. 30.	Died of an apoplectiform attack.
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{30}$ J. 1	Both 2½ mm., act to light, but very sluggish.	Died, '81, Oct. 31.	Died of diarrhœa.
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{35}$ J. 1	R. 3½ mm., L. 3 mm.; right very sluggish to light, left acts well; both act to accommodation.	Died, '82, Dec. 14.	
R. + 2 D. L. + 2 D.	R. $\frac{20}{15}$ J. 2 L. $\frac{20}{40}$ J. 4	Both 3 mm., regular; no action to light, but act well to accommodation.	Died, '82, Nov. 15.	
R. Emm.	R. $\frac{20}{25}$ J. 1 L. blind	R. 3½ mm., L. 3; both act to light and to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{25}$ J. 6 L. $\frac{20}{25}$ J. 2	R. 3 mm., L. 2¾, regular; act fairly well to light and well to accommodation.	Died, '82, Aug. 6.	
Emm.	R. $\frac{20}{30}$ J. 4 L. $\frac{20}{30}$ J. 6	R. 3 mm., L. 3½; both act to light, but right sluggish; both act to accommodation.	Died, '83, June 5.	
R. − 15 D. L. − 18 D.	R. $\frac{20}{7}$ J. 1 L. $\frac{20}{8}$ J. 1	R. 5 mm.; L. 4½ mm.; scarcely any action to light; act to accommodation.	Died, '82, Feb. 18.	Microscopical examination of optic nerves.
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{30}$ J. 1	Both 2¾ mm., regular; act to light and to accommodation.	Died, '83, Oct. 17.	Microscopical examination of optic nerves, &c. Plate I. Figs. 2 and 3. Pachymeningitis of cerebral dura mater.

of the main vessels, these parts presented perfectly normal characters.

TABLE I.—Showing the Condition of the Eyes in Sixt

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
13	C. B. *contd.*					fundus, the redness invading the physiological cups; edges decidedly blurred, though not equally so everywhere; outer edge of right pretty distinct for a short distance.
				1st	'82, Mar. 1	Much the same condition.
				„	„ May 3	Discs decidedly red, but less so than on former examinations—left the redder of the two; slight haziness of margins.
				2nd	„ Sept. 1	Much the same condition, but redness somewhat less marked.
				„	'83, Jan. 25	Right disc decidedly paler, but upper zone still of a distinct pink hue; edges very slightly blurred. Left disc still abnormally red; edges slightly blurred.
				„	„ April 26	Right disc pale—not pink; edges defined. Left disc full pink, edges defined.
				3rd	„ Aug. 22	Right disc greenish-grey throughout—no pink colour—distinctly atrophic in fact; vessels not diminished in size. Left disc, pale pink.
14	W. D.	M.	49	2nd	'81, Dec. 17	Discs normal. Choroidal atrophy around each, with patches of black pigment; a small recent hæmorrhage on outer margin of left; round right macula minute spots of choroidal atrophy. (Both lenses rather hazy.)
15	W. P.	M.	33	2nd	'81, Dec. 17	Both discs perfectly normal.
				„	'82, Mar. 8	„ „ „
16	W. K.	M.	28	2nd	'81, Dec. 21	Discs normal. Choroidal atrophy around each; and one or two spots of this at periphery of left fundus.
				„	'82, Mar. 11	Same condition.
17	J. L.	M.	46	2nd	'81, Dec. 11	Discs normal. A pearly patch of opaque fibres on inner side of right.
18	W. A.	M.	37	1st	'81, Dec. 25	Both discs perfectly normal.
				„	'82, Mar. 11	Both discs decidedly white, greyish white; inner edge of left alone somewhat pink.
				2nd	„ May 31	Right disc very pale, but slight tinge of pink on upper and inner side; left, lower and temporal portions very white, remainder pink.
				„	„ Aug. 23	Same condition.
19	P. M.	M.	42	2nd	'81, Dec. 26	Both discs perfectly normal (full red).
				„	'82, Mar. 8	Same condition.
				„	„ Dec. 28	„ „
				„	'83, Sept. 12	„ „
				3rd	'84, Feb. 20	„ „
20	W. Y.	M.	42	2nd	'81, Dec. 26	Both discs perfectly normal.
				„	'82, Mar. 8	Same condition.
				„	'83, Jan. 8	„ „
21	J. P.	M.	36	„	'81, Dec. 26	Both discs decidedly red—abnormally so; outer edge of left a little indistinct.
				„	'82, Mar. 11	Discs normal.

SIXTY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2½ mm., right acts very slightly to light, left acts freely; both act well to accommodation.		
Emm.	R. $\frac{20}{70}$ J. 16 L. $\frac{20}{30}$ J. 12	Both 3½ mm., right very slightly elongated vertically, very sluggish to light, but act slightly; act slightly to accommodation.	Died, '82, Mar. 8.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 3 mm., slightly elongated upwards and inwards; very sluggish action to light; act well to accommodation.	Died, '82, June 11.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	R. 3¼ mm., L. 3; R. acts well to light; L. sluggish; both act well to accommodation.	Died, '82, Nov 1.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2½ mm., regular; act to light and to accommodation.	Removed from asylum, '82, Nov. 2.	
Emm.	R. $\frac{20}{15}$ J. 1 L. $\frac{20}{20}$ J. 1 R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1 R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 3 mm., R. acts well to light, L. sluggish; both act to accommodation.	Removed from asylum, '82, Aug. 23.	The variations in the acuteness of vision, as tested by types at different periods, was doubtless due to the mental inattention that existed during the early stage of excitement.
Emm.	R. $\frac{20}{20}$ J. 6 L. $\frac{20}{20}$ J. 6	Both 4 mm., regular; act well to light and to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2 mm., regular; no action to light, but act well to accommodation.	Died, '83, Dec. 3.	
Slightly myopic.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2 mm., regular; no action to light, act well to accommodation.	Died, '82, Sept. 11.	

TABLE I.—Showing the Condition of the Eyes in Sixt

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
22	J. R.	M.	50	2nd	'81, Dec. 26	Discs normal (full red).
"				"	'82, Mar. 11	Same condition.
"				"	'83, Jan. 5	Right discs normal (pink); left, inner edge a little blurred.
23	E. J.	F.	35	3rd	'82, Jan. 2	Both discs somewhat white, but probably normal. (A slight opacity in left lens.)
24	W. R.	M.	43	3rd	'82, Jan. 6	Discs normal.
25	P. T.	M.	49	2nd	'82, Jan. 6	Discs normal (pink).
				"	" April 26	" "
				"	'83, Jan. 5	" "
26	E. C.	F.	37	1st	'82, April 12	Discs normal.
				"	'83, Oct. 28	" "
27	J. M.	M.	42	2nd	'82, April 26	Discs normal.
28	S. B.	F.	40	1st	'81, Feb. 12	Discs normal.
				2nd	'82, April 7	Both discs distinctly atrophic; greyish-white; punched out appearance of edges; vessels, both arteries and veins very small, particularly the former.
				"	" Aug. 2	Discs greyish-white; appear flat; no distinct cupping; vessels as before.
				3rd	'83, Jan. 25	Both discs of a staring greyish-white; edges sharply defined; vessels as before.
29	E. M.	M.	39	2nd	'82, April 29	Discs normal (pink).
30	T. W. C.	M.	35	2nd	'82, May 31	Discs normal (right, pink; left, red).
				"	" Dec. 28	" " " "
31	J. H.	M.	38	2nd	'82, May 31	Both discs normal (pink).
32	J. H.	M.	43	2nd	'82, June 11	Both discs normal (pink). Edges of left somewhat jagged, but not obscured.
				"	'84, Feb. 20	Same condition.
33	M. K.	F.	45	2nd	'82, June 14	Both discs pale; nasal sides pale pink, temporal sides whitish, and present a stippled appearance; choroidal disturbance on outer side of each (progressive). Some want of definition at outer margin of right disc.
				"	'82 Nov. 4	Discs appear normal (pink); edges defined. (Cups shelve off gradually on temporal sides; lamina cribrosa well seen in each.)
34	T. W.	M.	47	2nd	'82, June 14	Both discs very red throughout—hyperæmic; the redness is not simply due to diffused capillary tint, but there appears an actual development of minute vessels; margins a

SIXTY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
Emm.	R. $\frac{20}{15}$ J. 8 L. $\frac{20}{15}$ J. 8	R. 2½ mm.; L. 3 mm.; right, no action to light; left, very sluggish; both act to accommodation.	Died, '83, Oct. 15.	
..	..	R. 2 mm., L. 3½ mm.; scarcely any action to light, but act to accommodation.	Died, '82, Jan. 26.	Microscopical examination of optic nerves. Plate I. Fig. 1.
..	..	Both 3 mm.; act to light and to accommodation, but sluggish.	Died, '82, Mar. 11.	Indirect examination only. Large ulcers on cornea. Microscopical examination of optic nerves.
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{25}$ J. 1	Both 2½ mm., regular; act fairly well to light, and well to accommodation.	Died, '83, July 8.	
Emm.	R. $\frac{20}{15}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 3 mm.; scarcely any action to light.	Still under observation.	
R. Emm. L., simple myopic astigmatism.	R. $\frac{20}{15}$ J. 1 L. $\frac{20}{100}$ J. 1	Both 3 mm.; right acts well to light; left, no action; both act to accommodation.	Died, '82, April 26.	Accidentally suffocated by food getting into larynx. Microscopical examination of optic nerves.
..	..	.. ..	Died, '83, Mar. 14.	Microscopical examination of optic nerves. Plate II. Figs. 4 and 5.
	Could not count fingers accurately.	Both 3 mm., regular; very sluggish to light, but act to accommodation.		
Emm.	..	R. 2½ mm., L. 3, regular; scarcely any action to light, but act to accommodation.	Died, '82, Aug. 2.	
..	..	R. 3 mm., L. 2½; right slightly irregular; both act fairly well to light; very active to accommodation.	Died, '83, Sept. 9.	
..	..	R. 4 mm., L. 2½, regular; no action to light; act to accommodation.	Died, '83, June 23.	
R. + 2 D. L. + 2 D.	..	Both 3½ mm., regular; act well to light and to accommodation.	Still under observation.	
R + 3 D L + 3 D	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{30}$ J. 1	Both 1¾ mm.; no action to light; act well to accommodation.	Died, '82, Nov. 11.	Microscopical examination of optic nerves.
R + 1 D L + 1 D	R. $\frac{20}{30}$ J. 4 L. $\frac{20}{25}$ J. 4	Rt. 3 mm.; left 3½; regular; act well to light and to accommodation.	Removed from Asylum, '83, Aug. 27.	

TABLE I.—Showing the Condition of the Eyes in

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
34	T. W. *contd.*					little misty, especially at upper part; a fine white line crosses inner side of left disc; ? exudation. Pigmentary disturbance on outer side of each.
				2nd	'83, Jan. 8	Both discs red, but probably less so than when last examined; upper and inner edge of right disc is slightly ill-defined, but elsewhere edges are quite clear.
35	J. G.	M.	36	1st	'82, Aug. 24	Both discs normal (pink); edges well-defined.
				2nd	'83, Jan. 8	Both discs perfectly normal.
36	H. K.	F.	40	2nd	'83, April 25	Right disc palish; margins somewhat indistinct; vessels normal. Left disc flat, whitish; margins pretty distinct; vessels very small. Advanced condition of retinitis pigmentosa in left eye. Floaters in left vitreous. Cortical striæ in each lens, most marked in left.
37	G. O.	M.	59	1st	'82, Nov. 14	Right disc very red, but perhaps not abnormally so; edges defined. Left, large glaucomatous cap, surrounded by a narrow rim, which is probably formed by the sclerotic.
				„	'83, Jan. 11	Same condition.
				„	'83, April 26	Same condition.
				2nd	'84, Feb. 20	Same condition, but pulsation of arteries noted in right eye. Some increase of tension in both globes.
38	J. B.	M.	36	2nd	'82, Nov. 14	Right disc pale pink; vessels rather small; white tissue around vessels; edges of disc defined. Left disc very pale; vessels decidedly small; much white tissue around vessels. Edges defined.
39	W. R.	M.	28	1st	'82, Dec. 26	Right disc red throughout; outer margin quite clear and distinct; inner margin slightly softened, though edge can be traced; white tissue very conspicuous around some of the vessels, even the very small ones; a patch of white tissue around a central artery just at its bifurcation is very conspicuous. Left disc much the same condition, but tint is more pink than red; some slight blurring of inner margin; white lines along vessels distinct, but not so well marked as in right. Vessels well filled in each.
				2nd	'83, Sept. 12	Same condition.
				3rd	'84, Feb. 20	Right disc pink; inner margin a little indistinct (scarcely any physiological cup). Left, pink; inner margin a little indistinct. Vessels normal in each.
40	H. T.	M.	40	2nd	'82, Dec. 26.	Both discs rather red, left the more so; edges well defined; vessels normal.
41	G. Mc.G.	M.	49	2nd	'82, Dec. 28	Both discs full red, but redness does not invade physiological cups, and edges are defined.
42	C. A. W.	M.	37	1st	'83, Jan. 10.	Both discs decidedly red throughout; edges defined; white tissue around vessels.
				„	'83, April 26	Both discs very red throughout; redness striated; margins run into retina; a little white tissue around vessels.
				2nd	'83, Sept. 12	Much the same; redness very well marked, and margins of discs very indistinct to direct examination.

SIXTY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{25}$ J. 1	R. 3 mm., L. $2\frac{3}{4}$, regular; act well to light and to accommodation.	Died, '83, July 30.	
R. Emm. L – 5 D	..	R. $4\frac{1}{2}$ mm., L. 3, regular; scarcely any action to light, but act to accommodation.	Still under observation.	
..	R. $\frac{20}{50}$ J. 12 L. blind	..	Still under observation.	
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{20}$ J. 1	..	Died, '83, Feb. 24.	
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{25}$ J. 1	R. 4 mm., L. $4\frac{1}{4}$, regular; act very slightly to light, but well to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{25}$ J. 1	..	Died, '83, April 12.	Microscopical examination of optic nerves.
Emm.	..	..	Died, '83, May 23.	
..	..	Both 3 mm.; act to light and to accommodation.	Died, '83, Nov. 16.	Died of pneumonia. Microscopical examination of optic nerves, etc. Plate II. Fig. 6.

TABLE I.—Showing the Condition of the Eyes in

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
43	A. K.	M.	54	2nd	'83, Jan. 11	Both discs pale, right decidedly so; vessels both arteries and veins distinctly small.
44	P. W.	M.	38	2nd	'83, Jan. 13	Right disc of a well-marked grey colour throughout, distinctly atrophic; vessels distinctly diminished in size, especially the arteries; a little choroidal disturbance at upper and outer part. Left, of a pale grey tint throughout; vessels slightly diminished in size, especially the arteries.
				„	'83, April 26	Both discs very white—have a punched out appearance, and appear somewhat excavated. Vessels diminished in size.
45	J. W.	M.	40	2nd	'83, Jan. 28	Right disc very pale; vessels distinctly diminished in size. Left disc (seen only by indirect examination) appeared normal (pink). Both lenses very hazy, and in right a distinct peripheral flocculent opacity on outer side.
46	R. B.	M.	56	2nd	'83, April 28	Both discs normal (pink); vessels normal.
47	W. W.	M.	35	1st	'83, April 28	Both discs normal (pale pink); vessels normal.
				2nd	'84, Feb. 20	Same condition.
48	S. Y.	M.	29	2nd	'83, April 28	Both discs very pale, with a very slight tinge of pink; R. the paler of the two; edges very clear and defined. Vessels perfectly normal.
49	S. B.	M.	35	2nd	'83, April 28	Both discs normal (pink); vessels normal.
50	W. C.	M.	57?	2nd	'83, June 23	Both discs perfectly normal (pink).
51	R. N.	M.	50	1st	'83, June 23	Both discs apparently normal, but edges a trifle hazy, especially in left; colour of discs, pink. (?) Arteries somewhat small. Floaters in each vitreous.
				„	'84, Feb. 20	Both discs red; margins a little misty—feathery. Arteries distinctly small.
52	J. B.	M.	40	1st	'83, June 23	Both discs normal (pale pink); vessels normal. A small spot of pigment on posterior surface of L. lens.
				„	'84, Feb. 20	Same condition.
53	A. B.	M.	32	1st	'83, June 23	Right disc decidedly grey—greenish grey; edges very clear; slight hollowing; vessels normal. L. same condition, but a little less marked.
				„	„ Nov. 23	Much the same condition, but, if anything, more marked; L. is, perhaps, now a little the paler of the two.
				2nd	'84, Feb. 20	Both discs greenish grey throughout. Arteries distinctly small.
54	P. McG.	M.	52	3rd	'83, June 23	Both discs normal (pale pink). Lenses; muddy striæ at lower and inner part of periphery of each.

SIXTY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE *(continued)*.

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
..	R. $\frac{20}{25}$ J. 16 L. $\frac{20}{25}$ J. 16	..	Died, '83, June 3.	
Emm.	R. $\frac{20}{0}$ J. 19 L. $\frac{20}{0}$ J. 8	Both $1\frac{3}{4}$ mm., regular; no action to light, but act to accommodation.	Died, '83, May 5.	Died of pneumonia. Had clinical signs of locomotor ataxy, and after death grey degeneration of posterior columns of cord was found throughout greater portion of length. Optic nerves distinctly wasted to naked eye examination.
..	..	..	Died, '83, July 28.	Had clinical signs of lateral sclerosis.
Emm.	..	R. 3 mm., L. $2\frac{1}{2}$, regular; no action to light, act fairly well to accommodation.	Died, '83, July 29.	
Very slight myopic astigmatism.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{30}$ J. 1	R. 3 mm., L. 4; R. acts very slightly to light, L. not at all; both act fairly well to accommodation.	Still under observation.	
Emm.	..	..	Died, '83, Oct. 8.	
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{25}$ J. 1	R. 3 mm., L. $2\frac{3}{4}$, regular; do not act to light, but act to accommodation.	Died, '84, Jan. 8.	
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{25}$ J. 1	Both $2\frac{3}{4}$ mm., regular; act to light, but sluggish; act well to accommodation.	Removed from Asylum, '83, July 23.	
Emm.	R. $\frac{20}{25}$ J. 14 L. $\frac{20}{25}$ J. 14	Both $2\frac{3}{4}$ mm., regular; sluggish action to light; act well to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{25}$ J. 1 L. $\frac{20}{25}$ J. 1	Both $1\frac{3}{4}$ mm., regular; no action to light, but act to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{25}$ J. 6 L. $\frac{20}{25}$ J. 6	Both 3 mm., regular; no action to light, and very little to accommodation.	Still under observation.	Partial paralysis of 3rd nerve in right eye, indicated by external strabismus and slight ptosis.
..	..	..	Died, '83, Aug. 8.	

TABLE I.—Showing the Condition of the Eyes in

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
55	J. T.	M.	42	2nd	'83, June 23	Both discs normal (pink). R., well-marked appearance of opaque fibres surrounding the disc, the margins of which, however, are defined, the opacity not invading the surface of the disc itself. L., considerable pigmentary disturbance at inner and outer margins.
				„	'84, Feb. 20	Same condition.
56	T. H.	M.	42	2nd	'83, June 23	Both discs normal (pink).
				„	'84, Feb. 20	Same condition.
57	H. C.	F.	44	2nd	'83, Aug. 22	Both discs normal (pale pink); arteries rather small; choroidal disturbance around right; floaters in both vitreous bodies; conical cornea in left eye.
58	J. N.	M.	43	2nd	'83, Sept. 4	Both discs pale pink; edges defined; well-marked sclerotic rim, forming a small crescent on outer side; vessels a little small especially on left disc.
59	H. E.	M.	42	2nd	'83, Oct. 27	Both discs perfectly atrophic; surface flat, edges blurred; veins very slightly diminished in size, arteries considerably so.
				„	'84, Feb. 20	Same condition; veins much diminished in left disc. Numerous small pigment spots in retina.
60	R. B.	M.	36	2nd	'83, Nov. 23.	Both discs full red; inner edges slightly softened. R., well-marked crescent at outer and lower parts, and small one on inner. L., small crescent at outer and lower part.
61	M. A.	F.	45	2nd	'83, Dec. 8	Both discs normal (full pink).
				„	'84, Feb. 23	Same condition.
62	E. T.	F.	25	1st	'84, Jan. 19	R. disc, margins very indistinct; vessels diminished, the arteries being very fine; retinitis pigmentosa, the pigment extending quite up to disc; one or two floaters in vitreous. L. disc, and retina in same condition, but less marked; disc red in centre; the pigment extends only to the middle zone of retina.
63	M. C.	F.	38	1st	'84, Jan. 19	Both discs normal (red); vessels normal. A slight granular appearance at R. macula, and at L., pale buff patches finely sprinkled with black.
				„	„ Feb. 23	Same condition.
64	M. R.	M.	32	1st	'84, Jan. 20	Both discs pale pink; R. the paler of the two. A small, irregular-shaped crescent around each, especially at outer part. Middle zone of each fundus covered with well-marked circular, pigmented, white patches of choroidal inflammation, ? syphilitic, most marked in L.; floaters in L. vitreous.
65	W. W.	M.	52	2nd	'84, Jan. 20	Both discs normal (red).
66	S. F.	F.	23	3rd	'84, Feb. 2	Both discs normal (pink).

SIXTY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
R. −1 D L. −1 D	..	Both 3 mm., regular; act to light and to accommodation.	.. Still under observation.	
Emm.	..	..	Still under observation.	
R. + 2 D. L. + 3 D.	..	..	Died, '84, Feb. 22.	
..	..	Both 3 mm., regular; act to light, but sluggish; act well to accommodation.	Died, '83. Nov. 28.	
Emm.	Blind	Both 4 mm.; no action to light; act slightly to accommodation.	Still under observation.	Has signs of sclerosis of lateral columns of cord.
Slightly + each eye.	..	..	Died, '83, Dec. 7.	A case which ran a very rapid course.
Emm.	..	R. 2½ mm., L. 2. R. acts very slightly to light; L. not at all. Both act to accommodation.	Still under observation.	
Emm.	..	R. 3 mm., L. 3½. Both act to light, but sluggish; act to accommodation.	Still under observation.	R., well-marked nebula about centre of cornea; on anterior surface of lens, a cluster of small brownish spots. L., two faint nebulæ on cornea, one or two old adhesion bands, and an appearance as of new growth around margin of dilated pupil.
R. simple + astigmatism. L. Emm.	R. $\frac{20}{30}$ J. 4 L. $\frac{20}{30}$ J. 1	Both 3 mm., regular; act to light and to accommodation.	Still under observation.	
R. − 3 D. L. − 3 D.	..	..	Still under observation.	
..	..	R. 4 mm., irregular; L. 3, act slightly to light.	Still under observation.	
R. − 5 D. L. Emm.	..	R. 5 mm., L. 4; both act to light.	Still under observation.	Has signs of lateral sclerosis.

TABLE II.—Showing the Condition of the Optic Discs, &c., in Twelve cases of Mania, in which Slight (or Doubtful) Abnormalities were Detected.

No.	Sex.	Age.	Form of Mania.	Condition of Fundus, &c.
1	F.	38	Acute (recent)	Both discs pink, edges perhaps a little hazy; a spot of pigment on outer edge of right.
2	F.	53	„ „	Both discs very red, about the same colour as the fundus; edges a little blurred.
3	F.	42	„ „	Both discs red and hazy. ? Vessels a little small. Well-marked syphilitic choroiditis in each eye, all over fundus; gross changes; large white patches mottled with black.
4	M.	42	„ „	Both discs decidedly red, right the redder of the two, and the margin of this, especially at upper part, is not very clearly defined (probably on account of similarity of colour between it and fundus).
5	F.	23	Puerperal	Both discs perfectly normal; arteries rather small; *veins abnormally full* (a second examination made after an interval of 11 months, when patient was convalescent, showed the veins to be full, but not abnormally so; condition otherwise unchanged).
6	F.	29	„	1*st examination.*—Both discs full red; edges well-defined; striation of small vessels distinct. 2*nd examination* (12 days after 1st).—Much the same condition; substance of disc has a slightly softened appearance.
7	F.	30	„	*Examination 24 hours before death.*—Both discs red, but redness does not invade central cups; colour of left disc almost that of surrounding fundus, but edge can be traced, though it is somewhat indistinct.
8	F.	22	„	Both discs red throughout; upper and inner portion of left disc slightly misty. (Note.—*Patient had mitral stenosis.*)
9	F.	30	„	Both discs decidedly red, but edges are perfectly defined. (*Indirect examination only; patient very excited.*)
10	F.	41	Chronic	R. disc greyish-white; vessels decidedly small; enormous staphyloma around disc developed chiefly to outer side, extending by a series of spots of partial atrophy, as far as macula; small central opacity in posterior capsule of lens; numerous floating bodies, large and small in vitreous, which fall rapidly; −12 D. L. disc has a faint tinge of pink; vessels larger than in right; a small spot of atrophy in vicinity of macula; small floaters in vitreous; −7 D.
11	F.	65	„	Both discs normal (pink); vessels small, especially arteries (both lenses hazy, and marked cortical striæ in each).
12	F.	34	Delusional (monomania.)	Discs somewhat red, and edges a little indistinct.

TABLE III.—Showing the Condition of the Optic Discs, &c., in Nine cases of Melancholia, in which Slight (or Doubtful) Abnormalities were Detected.

No.	Sex.	Age.	Form of Melancholia.	Condition of Fundus, &c.
1	F.	46	Active (recent)	Discs normal (pale pink), *Arteries* full—as large as veins or nearly so. Pigmentary deposit on outer side of each disc.
2	F.	27	„ „	Discs decidedly red, but probably normal.
3	F.	55	Passive (recent)	Right disc palish, flat; margins a little indistinct; arteries small; left disc whitish; margins a little blurred; appears flat. Vessels, both arteries, and veins, decidedly small. [History of chronic alcoholism.]
4	F.	30	„ „	Both discs normal (pale-pink); vessels, both arteries and veins, rather small.
5	M.	46	„ „	Both discs pale, with a shade of pink. (Three examinations made at intervals of 3 or 4 months; same condition each time, except that on first examination, the arteries were thought to be a little small; they were however normal on subsequent examinations.) On anterior surface of capsule of right lens, very small brown pigment spots. [This case has some of the symptoms of General Paralysis, and that disease cannot at present be confidently excluded.]
6	F.	42	„ „	Both discs pale pink; edges slightly misty; vessels a little small; several recent hæmorrhages, small, and of moderate size, in both retinæ, not far from disc in right, especially about macula; one or two indistinct white patches in right retina, but there are several of these well marked in left. [Patient has chronic Bright's disease.]
7	F.	32	„ „	Both discs very pale; distinctly anæmic. [Patient masturbates excessively.]
8	F.	53	Passive (chronic)	Inner side of each disc, especially left, has a peculiar variegated or speckled appearance, due to the presence of five white lines or streaks; they otherwise appear normal; white lines very marked along some of the vessels. A pigmentary deposit at upper part of left fundus. (Two examinations made at an interval of 11 months—condition each time practically the same.)
9	F.	35	„ „	Both discs normal; a small horizontally-placed recent hæmorrhage on inner side of left disc.

TABLE IV.—Showing the Condition of the Optic Discs in Two cases of Mental Stupor during the Attack and after Convalescence.

No.	Sex.	Age.	——
1	F.	29	*1st examination—during condition of stupor.* Both discs perfectly normal (pink). *2nd examination—during lucid interval.* R. disc red, L. more pink than red; edges quite clear and defined; vessels normal. (Hypermetropic astigmatism in each eye.)
2	F.	40	*1st examination—during condition of stupor.* Both discs pale; edges well defined. *2nd examination—state of mental hebetude during convalescence.* Both discs pink, inclining to pale. *3rd examination—after convalescence.* Both discs still palish, as in last note.

TABLE V.—Showing the Condition of the Optic Discs, &c., in Fifteen cases of Dementia in which Abnormalities were Detected, the Majority being Slight or Doubtful.

No.	Sex.	Age.	Form of Dementia.	Condition of Fundus, &c.
1	F.	64	Secondary to Mania.	Both discs colour of fundus, but edges distinct, except so far as indistinctness results from similarity of colour between disc and choroid; vessels a little small. (Patient has granular kidneys.)
2	F.	39	„	Discs normal (pale); arteries rather small.
3	F.	27	„	R. disc very red throughout; edges a little ill-defined; a band of white tissue stretching across central part of disc in a horizontal direction. L. disc, red; very slight want of definition about edges; no white tissue; vessels in each well filled.
4	F.	22	„	Both discs rosy red; edges of left perhaps slightly hazy to direct examination; veins large and dark; patch of pigment on inner side of right disc, and a cluster of pigment patches surrounded by an irregular greyish-white patch in vicinity of R. macula, over one of which patches a small artery can be traced by direct examination.(−7 D. each eye.)
5	F.	32	„	Both discs very red; margins slightly misty (two examinations at intervals of 7 months; same condition each time).

TABLE V.—Showing the Condition of the Optic Discs, &c., in Fifteen cases of Dementia in which Abnormalities were Detected, the Majority being Slight or Doubtful (*continued*).

No.	Sex.	Age.	Form of Dementia.	Condition of Fundus, &c.
6	F.	31	„	Both discs very red, and edges appear somewhat ill-defined, especially inner edge of left; vessels normal.
7	F.	53	„	Both discs pearly white; in both, arteries exceedingly fine, threadlike, and few in number; veins also rather small. R., white spots at macula of fair size, also a few to outer side of macula; below and to outer side of disc is a punched-out spot of choroidal atrophy, with fine retinal vessel passing over it; smaller and fainter spots in the vicinity of this. L., minute white spots around macula. (Patient has chronic Bright's disease.)
8	M.	50	„	Both discs red throughout; the slightest possible softening of edges. (Two examinations at interval of 2 months; same condition each time.)
9	F.	28	Primary (recent and chronic).	Both discs normal; tint inclining to pale; arteries rather small.
10	F.	30	„ „	Both discs very red; on inner side the surface of each disc has a slight velvety appearance (most marked in left); edges perfectly clear and defined.
11	F.	28	„ „	Both discs very red; white lines very distinct along some of the vessels.
12	F.	49	Organic.	R. disc greyish, and slightly swollen at the lower part; vessels rather small; three large recent hæmorrhages on inner side of disc; a large, irregular-shaped patch of choroidal atrophy, about size of disc, on outer side of this. L. disc, swollen; inner edge cannot be seen, outer very faint—quite woolly; veins large, white lines around; a large recent hæmorrhage on inner side of disc. [Patient had chronic Bright's disease.]
13	M.	47	„	R. disc very red; some pigmentary disturbance around chiefly on outer side; Left disc, similar conditions but less marked. [Patient had R. hemiplegia and aphasia.]
14	F.	67	Senile	Both discs very red; slight crescent at outer side of each most marked in R.; lenses; hazy, with cortical striæ. (R. eye,—5 D.)
15	M.	61	„	Both discs pale pink; vessels a little small, especially the arteries; discs have a hollowed appearance; lenses; slight cortical opacities.

TABLE VI.—Showing the Condition of the Optic Discs, &c., in Seven cases of Insanity associated with Epilepsy, in which Slight (or Doubtful) Abnormalities were Detected.

No.	Sex.	Age.	Form of Mental Disorder.	Condition of Fundus, &c.
1	F.	28	Imbecility	Right disc distinctly hyperæmic in centre. (Left disc could not be examined owing to patient's restlessness.)
2	F.	17	„	Both discs normal (rosy-red); edges well defined; arteries a little small; veins full.
3	F.	20	„	Both discs very red, the redness invading slightly the physiological cups. Right eye a few scattered yellowish-white spots a little above macula; left, the same, but running into macula, and in addition two or three spots between macula and disc.
4	F.	37	„	Both discs very red—abnormally so; margins very slightly blurred; very well marked white lines along vessels—best seen in right disc.
5	F.	12	„	Both discs full red. On inner side of right disc, a distinct white streak.
6	M.	48	Slight Dementia.	Both discs normal (pink); arteries small.
7	F.	32	Profound Dementia.	Both discs pale, the right being the paler of the two; probably normal. (Two examinations at an interval of 3 weeks—same condition each time, except that on second examination, a minute hæmorrhage was noted on an artery on right side.)

DISTRIBUTION OF ANÆSTHESIA IN CASES OF DISEASE OF THE BRANCHES AND OF THE ROOTS OF THE BRACHIAL PLEXUS.

BY JAMES ROSS, M.D., LL.D., F.R.C.P.

THE following is the account which is usually given in anatomical works of the distribution of the cutaneous nerves

FIG. 1.

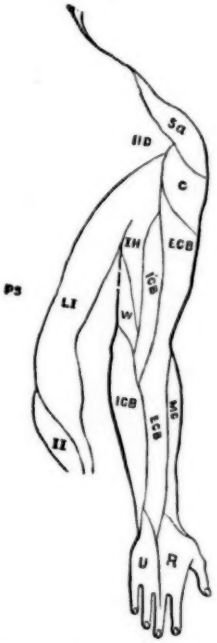
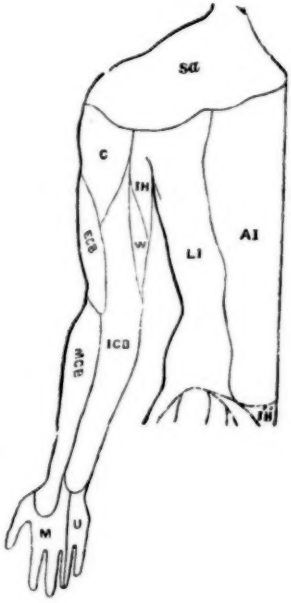


FIG. 2.



Figs. 1 and 2 (after Flower). Cutaneous nerves of the trunk and upper extremity.

of the upper extremity (Figs. 1 and 2). The inner surface of the arm and forearm, as well as the adjoining portions of

the anterior and posterior surfaces, are supplied by the intercosto-humeral (I H), the nerve of Wrisberg (W), the internal cutaneous (I C B), and branches of the ulnar (U). All these nerves are derived from the roots of the eighth cervical, and first and second dorsal nerves. The superior portion of the shoulder is supplied by the supra-clavicular and supra-acromial branches (S A) of the cervical plexus; and the inferior part of the surfaceof the shoulder, as far as the insertion of the deltoid, is supplied by branches of the circumflex nerve (C), which is most probably derived from the fifth cervical root. The outer surface of the arm and forearm, as well as the adjoining portions of the anterior and posterior surfaces, is supplied by the internal (I′ C B) and external (E C B) cutaneous branches of the musculo-spiral nerve, and the cutaneous branches of the musculo-cutaneous nerve (M C), the musculo-spiral and musculo-cutaneous nerves being most probably derived from the fifth, sixth, and seventh cervical roots. The back of the hand is usually said to be supplied by the radial (R) and ulnar (U) nerves, the radial supplying three and a half fingers, and the ulnar one and a half. The front of the hand is supplied by the median (M) and ulnar (U) nerves, the former supplying three and a half fingers, and the latter one and a half. The skin of the ball of the thumb is also supplied by branches of the musculo-cutaneous and radial nerves. The account which Krause gives of the distribution of the digital branches of the nerves of the hands differs in important particulars from that usually given in English anatomical works; and we shall immediately see that the former corresponds much better than the latter with the results of pathological observation. "Each finger," says Krause,[1] "is supplied with two palmar and two dorsal nerves, which are derived from the principal nerve trunks to the hand, and run along the radial and ulnar borders of the front and back of the fingers. The palmar are much larger than the dorsal branches; they run along the inner side of the palmar digital arteries to reach the tips of the fingers, and are chiefly

[1] Krause (Carl. F. T.), 'Handbuch der menschlichen Anatomie,' 3ᵉ Aufl. Hannover, 1879, s. 896. See also Henle, 'Handbuch der Nervenlehre des Menschen; Braunschweig,' 1871, s. 499.

distributed to the palmar surface. Opposite the first phalangeal joint of each finger, however, a long branch is given off on each side, which passes round the edge of the finger to the back of the second phalanx, whilst opposite to the second phalangeal joint a second smaller branch is given off on each side, which also passes round the finger to reach the back of the third phalanx. These branches divide, without forming a network, in the skin of the end of the finger and in that under the nail. The dorsal digital branches reach only as far as the dorsum of the first phalanges, where they form loops with the palmar branches, and it is only the branches which are distributed to the thumb alone that reach to the ungual phalanx" (Fig. 3).

FIG. 3.

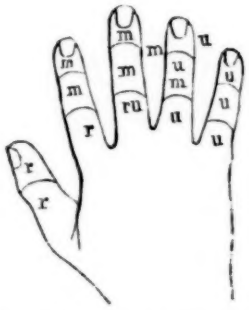


Fig. 3 (after Krause). Distribution of the Sensory Nerves on the back of the hand: r, radial; m, medium; and u, ulnar nerve.

It is now fully proved that when a nerve is divided, the fibres of the peripheral portion undergo rapid degeneration and lose their functional capacity. It might therefore be expected that when a sensory nerve is divided, the portion of skin to which it is distributed would become completely anæsthetic. But the experiments of Arloing and Tripier[1] on dogs and cats, have shown that in these animals the anæsthesia caused by section of a nerve by no means corresponds in extent to its area of distribution. With regard to the degree

[1] Arloing et Léon Tripier, "Recherches sur la sensibilité des téguments et des nerfs de la main." 'Archiv. de physiol. normale et pathologique,' tom. ii. Paris, 1869, pp. 33 et 307.

of anæsthesia caused by section of a cutaneous nerve, these experimenters found that section of one of the four nerve branches by which a digit of the dog or the cat is supplied, causes no recognisable diminution of its cutaneous sensibility, and that even section of two causes only slight general diminution of sensibility. Section of three branches, however, causes a very decided diminution of cutaneous sensibility, but complete anæsthesia is only established when all four branches are divided. Again, they found that on the fifth digit of the dog, which is supplied by branches from both the ulnar and radial nerves, partial anæsthesia only is caused by section of one or other of these nerves, but complete anæsthesia is caused by section of both nerves. In the cat, the fifth digit is supplied by branches of the ulnar nerve only, and consequently section of this nerve renders it completely anæsthetic. It is needless to enter into the details of these experiments; the general conclusion arrived at may be expressed by saying that when the four nerves of a digit are supplied from one only of the three nerves—the ulnar, the radial, and the median—which supply the skin of the paw, section of that nerve will render it completely anæsthetic; and when a digit is supplied from two nerves, both, and from three nerves, all three, must be divided before complete anæsthesia is established. This want of functional independence of the cutaneous nerves is easily explained by the free anastomosis which is known to take place, first, between branches of different nerves of considerable size, and second, between these nerves in the terminal networks described by Jacubowitsch and Beale. Arloing and Tripier have described a third method, by means of which nerve fibres from one nerve district may be distributed to another and far distant territory. After division of one of the branches of the median nerve of the dog or cat, for instance, they found that the animal gave manifest evidence of pain when the peripheral portion of the divided branch was pinched, and even division of the whole nerve did not entirely prevent these manifestations of sensibility. The peripheral portion of the divided branch was cut out and examined microscopically a few days after the section, and it was found that, although by far the greater number of the fibres were

degenerated, yet a few of them had remained healthy. A portion of the proximal end of the divided nerve was also examined microscopically, and a few degenerated fibres were discovered amongst a large number of healthy ones. From these observations, the authors conclude that the few fibres which remained healthy in the peripheral portion of the nerve still retained their connection with their trophic centres, while the few which were degenerated in the proximal portion were severed from their centres. As the trophic centres of these fibres must have been situated in the spinal ganglia or in the grey matter of the cord, they must have reached the divided branch from the peripheral side of the point of section, and have been pursuing a centripetal course, and the authors justly assume that these fibres conferred recurrent sensibility upon the peripheral part of the nerve.

It is not known how these fibres terminate, but Arloing and Tripier believe that they only ascend a short distance in the nerve, and it may be assumed that they are distributed to the skin and thus take an important part in supplying one district with nerve-fibres from neighbouring nerve-territories.

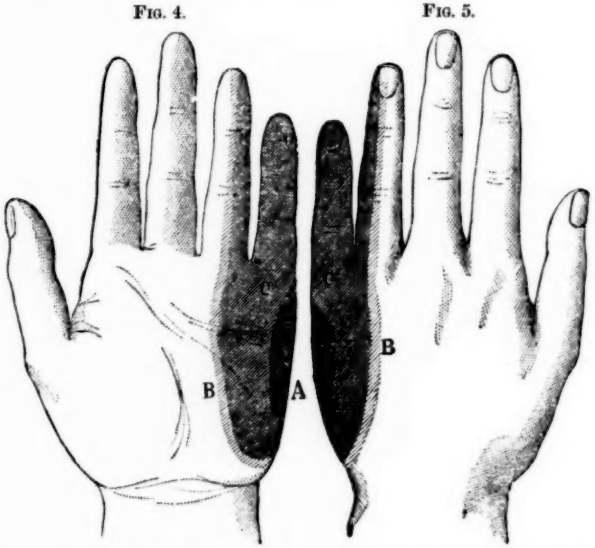
If we turn to pathological observation, it will be seen that in man the extent of the anæsthesia caused by division of a cutaneous nerve, either by surgical operation or injury, does not by any means correspond with the supposed anatomical distribution of the nerve.

In two cases of complete division of the ulnar nerve observed by Létiévant,[1] caused by a wound of the wrist, and in which all the muscles supplied by the nerve were completely paralysed and atrophied, the skin of the little finger and the inner half of the ring-finger, on their palmar and dorsal aspects, as well as that over the hypothenar eminence and the corresponding part on the back of the hand, was more or less anæsthetic. The least degree of anæsthesia was met with in a narrow border which formed the transition between the anæsthetic and healthy skin, and which is indicated in Figs. 4 and 5 by a light degree of shading (B). A patch of skin on the border of the hand near the hypothenar eminence, and which is indicated in Figs. 4 and 5 by the deepest shading (A), was completely anæsthetic, but in the remaining

[1] Létievant (E.), 'Traité des sections nerveuses.' Paris, 1873. (Obs. xxviii.) p. 70.

portion of skin, and which is indicated by the medium degree of shading, the sensibility was only considerably diminished.

In this case the extent of the anæsthesia corresponds pretty acurately with the supposed anatomical distribution of the cutaneous branches of the divided nerve, but although the nerve was completely divided at the wrist, the anæsthesia of even the little finger, which is supposed to be wholly innervated by the ulnar nerve, was incomplete.



Figs. 4 and 5 (after Létiévant). A, Deep shading, representing complete anæsthesia; B, light shading, represents slight, and C, medium shading, represents a medium degree of anæsthesia.

In another case,[1] reported by the same observer, a man aged 51 years had a wound of the wrist by a fall on a broken bottle, when he was only ten years of age. A deep cicatrix was visible above the wrist, and all the muscles of the hand supplied by the ulnar were completely paralysed and atrophied, but there was only a slight blunting of the sensibility in the region of distribution of the cutaneous branches of the injured nerve.

[1] Létiévant (E). Op. cit., Obs. xxix., p. 73.

This case appears to show that the anæsthesia caused by complete division of one of the cutaneous nerves of the hand diminishes in intensity with the lapse of time, and we shall hereafter find abundant evidence to show that it becomes diminished in extent also, and that the healthy territory gradually encroaches upon the affected district, so that the area of the latter becomes much restricted in the course of time.

The effects of division of the median nerve on the sensibility of the hand are no less interesting and instructive than those of injury of the ulnar.

FIG. 6.

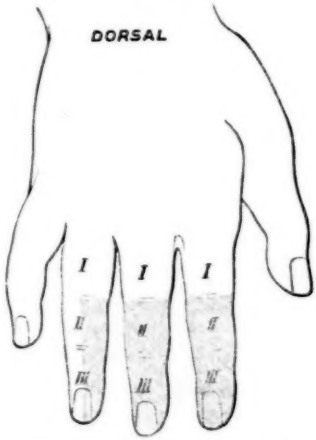


Fig. 6 (after Bernhardt), showing the distribution of the anæsthesia on the back of the fingers in a case of injury of the median nerve.

The case of a man aged 59 years, is reported by Bernhardt,[1] who sustained a fracture of the styloid process of the ulna, and severe concussion of the left forearm and hand through a fall. During the first three weeks after the injury no special treatment was adopted, and the patient was able to move all the fingers. At the end of this time the hand was placed in a plaster-of-Paris bandage which reached upwards as far as the middle of the forearm, and in which it was retained for three weeks. Four months after the injury, the patient came for the first time under the observation of Bernhardt. The wrist-joint was then moveable in all directions,

[1] Bernhardt, (M.) "Neuropathologische Beobachtungen." 'Archiv für Psychiatrie und Nervenkrankheiten,' Bd. v., 1875, p. 558.

but the muscles supplied by the median nerve were found paralysed and atrophied, while those supplied by the ulnar nerve were unaffected. On the palmar aspect of the hand the sensibility of the skin of the ball of the thumb and the radial half of the palm was considerably diminished, whilst that of the thumb, of the index, middle, and radial border of the ring finger was greatly diminished, the loss of sensibility becoming progressively greater from the basal to the ungual phalanges. On the dorsal aspect the sensibility of the skin of the thumb and little finger was intact over all the phalanges, but was much diminished over both borders of the middle and ungual phalanges of the index and middle fingers, and over the radial border of the corresponding phalanges of the ring finger (Fig. 6).

From five cases of division of the median nerve, carefully observed by Létiévant[1] he concluded that the anæsthesia is distributed over the palmar surfaces of the thumb, index and radial border of the middle finger, the thenar eminence, and the radial half of the palm, and over the dorsal surfaces of the ungual and middle phalanges of the index and middle fingers, and occasionally the ungual phalanx of the thumb. The anæsthesia is only partial except over the palmar surface of the whole of the ungual phalanx of the index finger, and part of the dorsal surface of the same phalanx. The different degrees of shading in Figs. 7 and 8 are intended to represent the varying degrees of anæsthesia, the very deep shading at A representing complete anæsthesia. It has already been remarked that the area of normal sensibility tends to encroach upon the anæsthetic area with the lapse of time; and in the following case, which came under my own observation, the area of anæsthesia resulting from division of the median was by no means so extensive on the dorsum of the fingers as in the cases reported by Bernhardt and Létiévant.

The case was that of a young man who was under the care of my colleague, Mr. Hardie, and who had received a deep wound on the front of the left forearm above the wrist, by falling on the broken edge of a glass bottle nine months before he came under my observation. The lower parts of the anterior surface of the left forearm presented a deep cicatrix which ran obliquely across the limb, and to which the tendons of the long flexors

[1] Létiévant, op. cit., p. 41.

of the fingers were adherent. The thenar and hypothenar eminences were flattened, and the hand was distorted into the "claw" form, which is so characteristic of division of the ulnar and median nerves. All the intrinsic muscles of the hand failed to re-act to either a strong faradic or a galvanic current, and it may consequently be concluded that no union had taken place in either the median or ulnar nerves. The distribution of the anæsthesia is shown in Figs. 9 and 10, but I much regret that the careful notes which were taken at the time have been mislaid, so

FIG. 7.

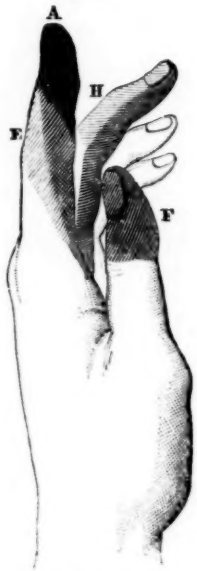
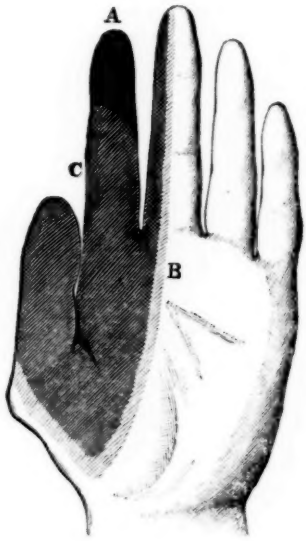


FIG. 8.

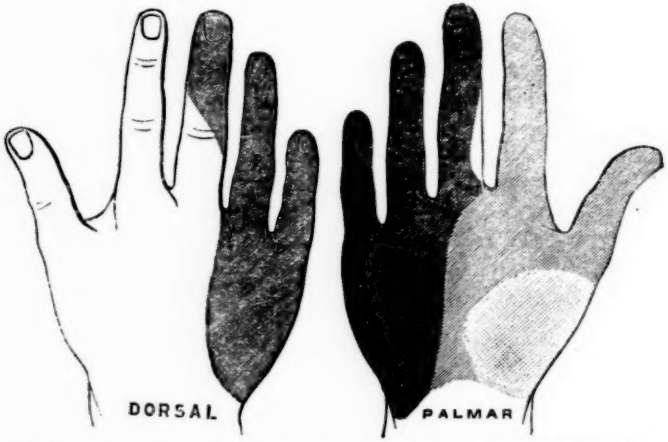


Figs. 7 and 8 (after Létiévant). Radial border and dorsal aspect of the hand showing the distribution of anæsthesia in a case of division of the median nerve. A, Deep shading representing more or less complete anæsthesia; B, E, and H, light shading, and C, medium shading, representing a slight and medium degree of anæsthesia respectively.

that I am unable to make any very positive assertions with regard to the diminution of the various forms of sensibility. The diagrams which were taken at the time, may be relied upon to represent accurately the extent of the anæsthetic area, and the varying degrees of the shading also represent in a general way the different degrees of anæsthesia which was present.

A very important case is reported by Dr. Weir Mitchell,[1] in which a portion of the musculo-spiral and median nerves were excised, at different times, for the relief of intense brachial neuralgia in a lady. Towards the end of 1871 Dr. Sapolini, surgeon to the King of Italy, removed an inch of the musculo-spiral nerve in the upper arm. The patient was astonished at the slight diminution of tactile sensibility which had followed the operation, but the relief obtained was only of short duration, inasmuch as the pain recurred eight days afterwards. The extensor muscles remained paralysed for some time, but motor power gradually returned in them about six months after the nerve was divided, and two years after the operation, voluntary power over the extensors was scarcely impaired. The sensory

FIG. 9. FIG. 10.



Figs. 9 and 10. Dorsal and Palmar aspects of the Hand, showing the distribution of anæsthesia in a case of division of the ulnar and median nerves. The different degrees of shading show in a general manner the degrees of anæsthesia present.

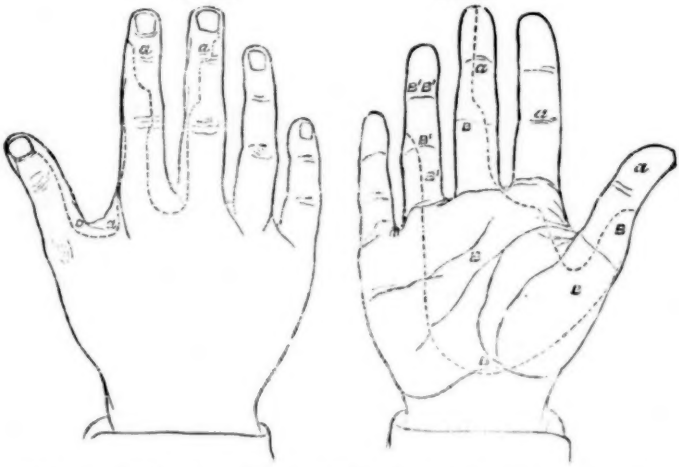
disorders present thirteen months after the operation, when the lady first came under Dr. Mitchell's care, are thus described:[2] "The radial and median sensation in the arm is normal, as well as that of the musculo-cutaneous nerve. In the radial area in the

[1] Mitchell (S. Weir). "Traumatic neuralgia; Section of Median Nerve." 'The American Journal of the Medical Sciences,' vol. ii., 1874, p. 17.

[2] Mitchell. Loc. cit., p. 19.

back of the thumb and first and second fingers, touch is referred correctly, and is everywhere felt: but throughout this region every touch is felt as both touch and pain. In a less degree this is true of certain parts of the whole arm, and most notably of parts just around the scene of the operations on the musculo-spiral. The ulnar territory is in all respects healthy by compass point. The sensitiveness of the thenar eminence is not acute, but increases as we approach the index-finger. The hyperalgesia is exquisite in the face of the second finger, and the radial side of the third finger; on the face of the thumb and the palm; over the first joint of the index. In these regions touch is so much more distinctly felt as pain that the degree of health of the power

FIG. 11. FIG. 12.

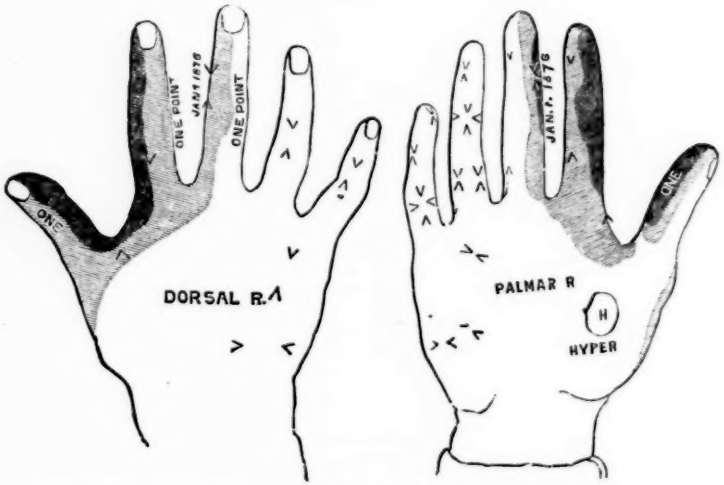


Figs. 11 and 12 (after Mitchell). Dorsal and Palmar aspects of the Hand respectively; *a*, pain and touch lost; B, touch lost; B′, touch slightly lessened.

of touch cannot be thoroughly studied." It is manifest from this description that not much anæsthesia had remained as the result of this operation, and the return of motor power in the extensors rendered it probable that union had taken place between the ends of the divided nerve. In 1873, fifteeen months after the first operation, three-quarters of an inch of the median was, in accordance with Dr. Mitchell's advice, excised by Dr. Brinton, and in order to prevent reunion the lower end of the nerve was doubled upon itself. The distribution of the anæsthesia to pain and touch observed at the end of two weeks is shown in Figs. 11

and 12. When the patient was examined nine months after the operation the area of anæsthesia had become considerably diminished on the palm, but remained nearly the same on the dorsal aspect of the hand. After a time the pain returned in the territory of the musculo-spiral nerve, and Dr. Brinton, still acting under Dr. Mitchell's advice, removed nearly three inches of the nerve on a level with Dr. Sapolini's operation, which had been performed two years and seven months previously.

FIG. 13. FIG. 14.



Figs. 13 and 14 (after Mitchell). Dorsal and Palmar aspects of the Hand. The light shading indicates the area in which tactile sensibility was diminished; the deep shading the area in which it was lost. The marks (> <) indicate the appreciation of separate points. A single (V) mark indicates that the points are not felt as separate. The absence of the (V) mark indicates complete loss of tactile sensibility. H, a small area in which there is hyperalgesia of the skin.

The condition of the sensibility on the dorsal and palmar aspects of the hand, four weeks after the operation, is represented in Figs. 13 and 14 respectively. A small spot of hyperalgesia (Fig. 14, H) was observed on the thenar eminence for which it is difficult to find an adequate explanation. The precautions taken to prevent reunion of the median, taken

[1] Mitchell (S. Weir). "Neurotomy." 'The American Journal of Medica Sciences,' vol. i., 1876, p. 322.

along with the fact that the muscles supplied by it below the point of division remained permanently paralysed, renders it extremely unlikely that its ends had become united, and no one can believe that the musculo-spiral nerve had become united four weeks after three inches of it had been excised, and consequently the terminal branches of the ulnar, and a few filaments from the internal cutaneous and musculo-cutaneous nerves had sufficed in this case to bestow sensibility upon the whole of the palmar and dorsal aspects of the hand with the exception of the comparatively small areas which are shaded in Figs. 13 and 14. The areas which are most deeply shaded in the figures, represent the regions of greatest anæsthesia, but even in these sensation was not entirely abolished, as the prick of a needle was still felt.

The disorders of sensibility caused by division of the musculo-spiral nerve are well illustrated in the case of a young soldier reported by Létiévant,[1] who had the nerve divided as the result of a wound by a poignard in the inferior and internal aspect of the forearm.

FIG. 15.

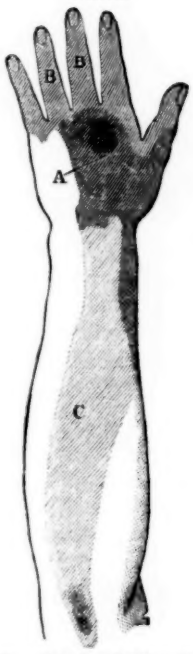


Fig. 15 (after Létiévant). Showing the distribution of the anæsthesia in a case of division of the musculo-spiral nerve in the arm. A, Deeply shaded, to show the area of greatest anæsthesia; B, shaded in a medium degree, to show the area of partial anæsthesia; C, faintly shaded, to show the area of slight anæsthesia.

The distribution of the anæsthesia on the back of the forearm and hand, two and a half years after the injury is represented in Fig. 15.

The highest degree of anæsthesia was found in a patch of skin over the back of the hand, and first phalanx of the thumb, and is represented by the deep shading (Fig. 15, A). A

[1] Létiévant (E.) Op. cit. (Obs. xxxix.) p. 96.

slighter degree of anæsthesia, represented by the medium shading in Fig. 15, B, was observed over the dorsum of the ungual phalanx of the thumb, and the back of all the fingers including the little finger, which is supposed to be innervated by the ulnar nerve. A still slighter degree of anæsthesia, represented by the light shading (Fig. 15, C), was found on the back of the forearm in the area of distribution of the external cutaneous branch of the musculo-spiral nerve.

The anæsthesia is widely distributed in this case over the back of the hand and fingers, and offers in that respect a marked contrast to the very limited area of anæsthesia in the case of resection of the median and musculo-spiral nerves reported by Dr. Weir Mitchell (Figs. 13 and 14). Another case of resection of a portion of the radial nerve is reported by Dr. Mitchell[1] in which the area of anæsthesia was of equally limited extent with his first case, although the localisation of the affected skin differed considerably in the two cases.

FIG. 16.

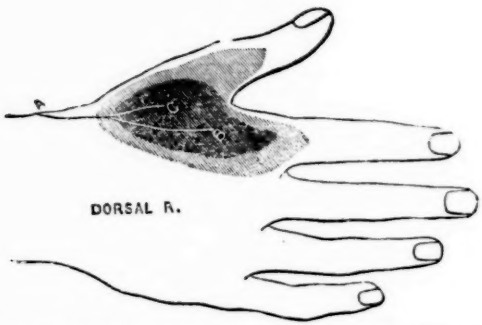


Fig. 16 (after Mitchell). The dorsal aspect of the hand two weeks after resection of the radial nerve. A B, and A C, the length of the portion removed. The light shading represents the area in which tactile sensibility was diminished; the deep shading the area in which it was lost.

In this second case, two inches of the radial nerve was excised by Dr. Hodge in the back of the forearm, for the relief of long continued and persistent pain in the back of the hand. In about six weeks the pain gradually returned, and tactile sensation, at first lost, also gradually reappeared. It was evident that the

[1] Mitchell (S. Weir). Loc. cit., p. 328.

nerve had become reunited, and about ten months after the first operation three inches of the nerve were removed, and the end of the distal extremity was folded so as to prevent reunion. The distribution of the anæsthesia two weeks after the second operation is represented in Fig. 16.

When the lesion of the musculo-spiral nerve is situated in the axilla, as in crutch paralysis, a certain degree of anæsthesia may be discovered in the anterior aspect of the forearm in the region of distribution of the superior branches of the external cutaneous nerve, and the internal cutaneous branch of the musculo-spiral, but loss of sensibility is never a marked feature of such cases. Division of the musculo-cutaneous nerve gives rise to partial anæsthesia along the radial border of the forearm, but excision or division of the circumflex nerve does not give rise to any manifest disorders of sensibility.

Sensory disorders in disease of the roots of the brachial Plexus.—Our data for determining the respective connections of the sensory nerves of the upper extremity with the cervical portion of the spinal cord are as yet very scanty. In the following case of *pachymeningitis cervicalis* at present under my care in the Barnes Convalescent Hospital, Cheadle, and in which the lesion is situated, as we shall find reason to believe, at the junction of the cervical and dorsal regions of the cord, the distribution of the anæsthesia in the upper extremities is very instructive.

Edward D——, aged 30 years, was admitted into the Manchester Royal Infirmary on October 16th, 1882, under the care of Dr. Ross.

The patient has always enjoyed good health; and, with the exception of an abscess of the testicle from which he suffered two years ago, and which troubled him for about five weeks, he has not had any illness worth mentioning up to the date of the present attack. The patient has led an unsteady life; he has many times slept, whilst in a state of intoxication, on the pavement, and has frequently gone to work during the day without changing his clothes, although he had been drenched on the previous night. His occupation is that of a dyer, but no evidence of chronic metallic poisoning can be discovered. He has not had syphilis, and no hereditary tendency to any particular disease can be found from his family history. About five months ago he began to suffer for the first time from pain between the shoulders, and at

the back of the neck, which was much increased by coughing, and by movement. The pain gradually increased in intensity until it became almost unbearable; it was "stabbing" in character, and so much worse at night, and in the early morning, that he had to pass many sleepless nights. The patient had the full use of his limbs and did not feel pain or any abnormal sensation in them until about five weeks ago. At that time he felt "prickling" sensations in his feet; these gradually travelled upwards until at the end of eight or ten days, they were felt over the whole of the lower extremities and body, up to the level of the first ribs, as well as down the inner borders of the arms, forearms, and the inner halves of the hands. A feeling of numbness soon followed the prickling sensations; this feeling also began in the feet and travelled upwards until, at the end of another eight or ten days, all the parts in which the prickling sensations were previously felt had become numb and insensible to pinching. The numb feeling was accompanied by a gradual loss of power of the lower extremities, so that the patient was soon unable to walk, and he likewise observed that his grasp was feeble. He was also troubled with a severe pain in the form of a belt passing round the abdomen, and with jerkings of the lower extremities, the latter being most troublesome at night.

Present Condition.—The patient lies on his back in bed in a helpless condition. The lower extremities are completely paralysed, passive movements of them provoke muscular tension, the cutaneous reflexes are increased, the patellar-tendon reactions are exaggerated, ankle clonus can be readily elicited on both sides, and the electrical reactions of the paralysed muscles are normal. The patient is unable to close either hand, and when he endeavours to grasp an object, the hand becomes hyperextended at the wrist, so that it is nearly at right angles to the forearm; the first phalanges are at the same time extended upon the metacarpal bones, while the second phalanges are flexed upon the first, and the first upon the second. The metacarpal bone of the thumb is abducted and drawn backwards, so that it is almost upon a level with the metacarpal bones of the fingers, and the phalanges are extended upon one another, and upon the metacarpal bone. The thenar and hypothenar eminences are flattened, the metacarpal bone of the index finger can be felt immediately under the skin, owing to the wasting of the abductor indicis, and when the mass of tissue which in health lies between the metacarpal bones of the thumb and index finger is grasped between the finger and thumb, it is felt to consist of scarcely anything more than a fold of skin, owing to the wasting of the abductor pollicis, and the flexor brevis pollicis.

The small muscles of the hands contract to a strong faradic current: they contract to the galvanic current with twenty-five cells Leclanché on negative closure, and with thirty cells on positive closure. There is incomplete anæsthesia of the skin of the lower extremities and trunk up to the level of the first rib, and the anæsthetic and sensitive areas merge into one another without being separated by a band of hyperæsthesia. The sense of temperature is almost entirely lost over the lower extremities and trunk, and two points have, on an average, to be removed four inches from each other before they are recognised as separate. The sense of pain as tested by pricking with a pin is not much diminished, but on pinching a fold of skin, the patient states that it feels numb; there is no perceptible retardation of sensibility, touch is localised fairly well, and the muscular sensibility is normal. The inner surfaces of the arm and forearm, and the inner half of the dorsal and palmar aspects of the hand and two and a half fingers, are also incompletely anæsthetic on both sides (Fig. 17). In these regions the patient can feel the prick of a pin fairly well, but he can bear strong pinching of the skin without giving evidence of pain. He cannot localise touch well in the anæsthetic areas, and two points have to be separated four inches before they are recognised as separate. The ordinary breathing of the patient is diaphragmatic, but he can still elevate the ribs by a voluntary effort, and the intercostal muscles are then seen to act. He has to strain a good deal before he urinates, but he has had no retention. The bowels are somewhat constipated. The pupils are widely dilated, but they react fairly well to light and accommodation. The optic discs are normal. All the internal organs are healthy.

April 11*th*, 1883.—The patient still lies on his back in bed, without having undergone much change since last report, although his general condition has on the whole improved. The right pupil is now considerably more contracted than the left, and the palpebral aperture is decidedly narrower on the right than on the left side.

Dec. 14*th*, 1883.—The patient's health had improved so much that he was transferred to the Cheadle Convalescent Hospital, about the middle of October last, and since that time a still further improvement has taken place. The anæsthesia has almost entirely disappeared; the small muscles of the hand have become plumper; the patient's grasp is good; and he can move his legs freely in bed, although muscular tension can still be provoked by passive movements of them, and the tendon reactions are somewhat exaggerated. The pupils are now equal, and there is no difference in the size of the palpebral fissures. The patient can raise himself in bed and

sit up, but is yet unable to walk without support. There is no prominence or thickening of the spinous processes of any of the vertebræ, and no tenderness on percussion of any part of the vertebral column.

Fig. 17.

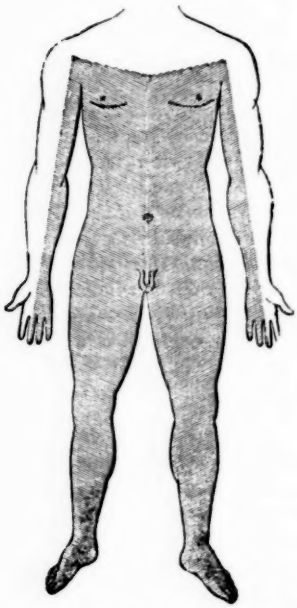


Fig. 18.

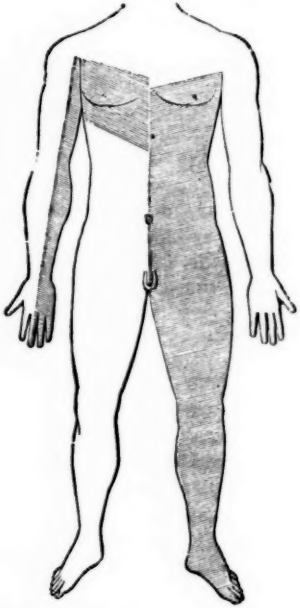


Fig. 17. The shaded portion shows the distribution of the anæsthesia in a case of cervical pachymeningitis, of which the lesion was situated on a level with the eighth cervical and first and second dorsal nerves.

Fig. 18. The shaded portion shows the distribution of the anæsthesia in a case of hæmatomyelia, in which the upper limit of the lesion was on a level with the eighth cervical nerve.

In this case, each hand assumed the peculiar claw-like form described by Charcot[1] as occurring in cervical pachymeningitis when the lesion is situated opposite the last cervical and first dorsal vertebra, and the distribution of the motor paralysis as

[1] See Charcot (J. M.), 'Leçons sur les maladies du système nerveux,' tome ii. 1877, p. 251.

well as the oculo-pupillary phenomena present in this case sufficiently indicated that the lesion was situated on a level with the junction of the cervical and dorsal regions of the cord.

The portion of skin which was anæsthetic in each upper extremity is supplied by the intercosto-humeral, the nerve of Wrisberg or small internal cutaneous, the internal cutaneous, and the sensory branches of the ulnar nerve. It is known from dissections that these nerves are derived from the eighth cervical, and first and second dorsal nerves, and these are the roots of the plexus which we would expect to have been involved in disease from the position of the lesion. It is scarcely necessary to add that the anæsthesia of the trunk and lower extremities were, in this case, caused by disease of the sensory conducting paths. In a case of hæmatomyelia recorded by myself,[1] the chief lesion was situated in the right lateral half of the spinal cord, and from the character of the motor, sensory and oculo-pupillary symptoms present it must have extended from the level of the ninth dorsal up to that of the eighth cervical nerve inclusive. The distribution of the anæsthesia is shown in Fig. 18. The anæsthesia of the inner half of the cutaneous surface of the upper extremity and of the belt which encircles the half of the thorax on the right side is to be explained by supposing that the hæmorrhage had destroyed on that side, either partially or wholly, the posterior grey horns on a level with the eighth cervical and upper nine dorsal nerves. The anæsthesia of the half of the trunk and lower extremity on the left side was doubtless caused by injury of the sensory conducting paths in their way through the cord after crossing over to the right side. If these cases do not teach us anything very new, they at least confirm the knowledge already acquired from dissections of the brachial plexus and experiments on animals, and they serve to illustrate in a striking manner the distribution of the anæsthesia caused by disease of the inferior roots of the brachial plexus and the second dorsal nerve.

In order to determine the distribution of the sensory disorders caused by disease of the superior roots of the

[1] Ross (J.), 'The Practitioner,' Sept. 1882, p. 172.

brachial plexus, we naturally turn to the cases of combined paralysis of the deltoid, biceps, brachialis anticus, and supinator longus muscles first described by Erb,[1] and in which the lesion is most probably localised in the root derived from the fifth cervical nerve. In these cases patients have complained of formication and numbness in the outer surface of the arm and forearm, as well as in the thumb and index finger,[2] but the sensory disorders are never prominent and soon disappear. It would, therefore, appear that the fifth root of the brachial plexus supplies sensory fibres to the external cutaneous and radial branches of the musculo-spiral, as well as to the musculo-cutaneous and probably also the median nerves, but the absence of anæsthesia and the rapid disappearance of the paræsthesiæ in the area of distribution of these nerves seem to indicate, that all of them receive sensory fibres from other roots of the plexus as well.

The following case of rupture of the brachial plexus, which was kindly sent to me by my friend Dr. Cowan, of Newchurch, may be worth reporting here, inasmuch as it will serve to illustrate the sensory disorders which are caused by rupture of all the roots of the brachial plexus with the exception of the communicating branch from the fourth nerve.

J. F., aged 17 years, entered the Manchester Royal Infirmary on December 2nd, 1882, under the care of Dr. Ross.

He never had any serious illness until six months before admission. Working in a cotton-mill, his left arm was caught between the strap and a revolving wheel. He was lifted from the ground and carried round by the wheel, until he fell on the opposite side. He was at first stunned by the fall, but on recovering he felt as if his right arm were dead; and it was found to be quite powerless, as it has since remained.

Present Condition.—The patient is a healthy-looking young man, fairly well built and free from organic disease of the internal organs. His left arm hangs powerless by his side when it is not

[1] Erb (W.), 'Verhandlungen des naturhistorischen Vereins zu Heidelberg,' 1875, s. 130; see also Ziemssen's 'Encyclopædia of the Practice of Medicine,' vol. xi., 1876, p. 561.

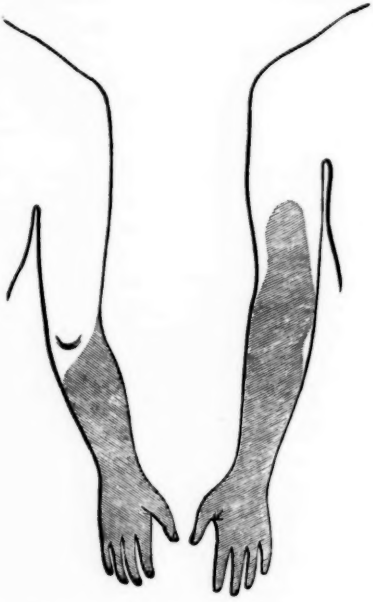
[2] See Hoedemaker (H. ten Cate), "Ueber die von Erb zuerst beschriebene combinirte Lähmungsform, an der oberen Extremität." 'Archiv für Psychiatrie und Nervenkrankheiten,' B. ix., 1879, s. 738.

held in a sling. All the muscles of the upper arm, forearm, and hand are completely paralysed and atrophied. The circumference of the left arm about its middle is 6½ inches, and of the right 8⅛ inches; and the circumference of the left forearm a little below the elbow is 7 inches, and of the right 9½ inches. The electrical reactions of all the nerves of the brachial plexus are lost. The faradic contractility of the affected muscles is also abolished, but the galvanic contractility is increased, while a stronger reaction is obtained from anodal than from cathodal closure. The latissimus dorsi and the lower two-thirds of the pectoralis major muscles are also paralysed and atrophied, but the upper third of the pectoralis major and the pectoralis minor as well as the internal and external rotators of the humerus, are unaffected. The serratus magnus on the left side is not paralysed, but when the paralysed arm is raised vertically by the observer and fixed in that position, the digitations of the muscle do not become so prominent on the left as on the right side during deep inspiration, and there appears also to be a slight degree of relative wasting of the left muscle, but the electrical reactions are normal. When the chest is at rest after the deepest expiration, the left half on a level with the nipple measures 14 inches and the right 14¾ inches; whilst after the deepest inspiration the left measures 14¾ inches, and the right 16½ inches. The patient can perform certain movements at the shoulder joint with the muscles that remain active, which might lead one to suppose, without careful examination, that the deltoid, triceps, and biceps possess a slight degree of motor power. By leaning his body well to the healthy side and somewhat backwards, he can raise the left arm above the horizontal level in a forward direction. While in this position he can extend the forearm upon the arm at pleasure. It is found, however, that the arm is raised partly by a powerful rotation of the scapula, during which the inferior angle is drawn well forwards and the superior external angle is well tilted upwards, and partly by the action of supra-spinatus muscle. Professor Alexander Ogston examined the patient along with me a few days ago, and both of us were convinced that there was no contraction of the deltoid during this movement. Extension at the elbow is produced by a powerful outward rotation of the humerus while the limb is held horizontally, this movement placing the forearm in such a position that it becomes extended by its own weight. And again, when the patient produces an inward rotation of the humerus the forearm becomes flexed by the action of gravity, and the hand moves towards and nearly reaches the patient's mouth.

Every form of sensibility is lost in the skin of the hand, and in

that of the forearm, with the exception of a small portion adjoining the elbow in its posterior and internal aspects. The skin of the inferior half of the anterior surface of the arm is also completely anæsthetic; but the superior half of the anterior surface, the skin of the whole of the external, posterior, and internal surfaces of the arm is sensitive to the prick of a pin, to pinching, and to touch, although those portions of skin manifest a slight diminution in the appreciation of separate points as compared with the corresponding

FIG. 19. FIG. 20.



Figs. 19 and 20. Posterior and anterior aspect of the arm respectively, in a case of rupture of the brachial plexus, the shaded area showing the distribution of the anæsthesia. By an oversight, the right arm is represented as being affected instead of the left.

parts of the healthy arm. Separate points are discriminated on the inner surface of the left or diseased arm above the elbow at a distance of 3 mm., on the healthy arm at 2 mm.; on the left arm near the axilla at 6 mm., on the right at 4 mm.; on the left arm

over the shoulder at 5 mm., on the right at 2 mm.; on the posterior surface of the left forearm near the elbow at 2½ mm., and on the right at 1¾ m. The distribution of the area of complete anæsthesia is indicated by the shaded portions of Figs. 19 and 20.

The sensitive area and that of absolute anæsthesia are separated by a band of about an inch in vertical extent, in which pricking with a pin and strong pinching are slightly felt, but on the whole, the transition from one region to another is comparatively abrupt.

The skin in the anæsthetic area is thin and smooth; the affected extremity becomes cold, and of a dark blue colour on exposure; and the pulse is even more perceptible on the affected than on the healthy side, depending most probably upon a wasting of the tissues on the diseased side, which causes the artery to feel as if it lay almost immediately beneath a thin covering of skin.

The left pupil is smaller than the right in a diffused light, and the difference between them becomes greater when the eyes are shaded. The left pupil is dilated to a medium, and the right to a maximum degree of dilatation by atropine. The palpebral fissure is decidedly narrower, and the eye appears smaller on the left than on the right side. The colour of the face on the two sides is the same, and the temperature, taken in the external meatus of the ear, was found on an average of several observations to be ·8° Fah. higher on the left than on the right side; in one observation the temperature was slightly higher on the right than on the left side, and in the last observation made there was no difference between the two sides.

Dec. 10.—A quarter of a grain of pilocarpine was injected subcutaneously. In a few minutes the body was bathed in perspiration, with the exception of the paralysed arm which remained quite dry. The left half of the face perspired almost as freely as the right half.

Aug. 1883.—Through the kindness of Dr. Cowan, I was enabled to show the patient at the meeting of the British Medical Association at Liverpool, where he was examined, amongst others, by Dr. Broadbent.

In the case which has just been reported, the presence of complete paralysis with atrophy of all the small muscles of the hand and of the flexors of the forearm showed that the motor fibres of the inferior roots of the brachial plexus were ruptured, while the presence of a similar affection of the deltoid, biceps, brachialis anticus, and supinator

longus showed that the motor fibres of the fifth root were severed, and consequently there are no grounds for believing that the intermediate roots derived from the sixth and seventh nerves had escaped, even although it is not accurately known what muscles are paralysed by isolated disease of one of those nerves. The absence, however, of paralysis of the inward and outward rotators of the humerus would seem to indicate that the motor fibres derived from the communicating branch to the brachial plexus from the fourth nerve had remained unaffected. But if the motor fibres of the roots of the plexus derived from the fifth, sixth, seventh, and eighth cervical, and first dorsal nerves were completely ruptured, it may be inferred that none of the sensory fibres of those roots had escaped, and the absence of electrical reactions in the affected muscles is totally opposed to the supposition that any reunion had taken place. The intercosto-humeral nerve was doubtless spared,[1] and it may be supposed to have conferred a degree of sensibility on the inner surface of the arm and a small part of the inner surface of the forearm which was only slightly below the normal as tested by a comparison with the corresponding parts of the healthy arm. The sensibility in the superior two-thirds of the anterior surface of the shoulder and arm, and on the whole of the external and posterior surfaces of the shoulder and arm as well as a small portion of the adjoining surface of the forearm was only slightly diminished as compared with corresponding parts of the healthy side, and sensation must have been conferred on these portions of skin by the sensory fibres of the communicating branch of the fourth nerve, and the sensory branches of the cervical plexus which descend over the shoulder.

In a case of excision of the brachial plexus by Maury and Duhring[2] for the relief of painful neuroma of skin, the sensations of pain and touch were absent, six months after the operation, over the whole of the skin of the forearm and hand as well as in that of the anterior aspect of the upper arm, as represented by the area

[1] See Paget (J.). 'The Medical Times and Gazette,' March 26, 1864.

[2] Maury and Duhring. "Case of exsection of the brachial plexus of nerves for the relief of painful neuroma of the skin," 'The American Journal of the Medical Sciences,' vol. ii., 1874, p. 29.

included within the lines *a a*, and *b b*, Fig. 21. The whole of the posterior, external, and internal surfaces of the arm were sensitive to touch, and to the prick of a needle, the sensation being more acute in the superior than the inferior part of the arm. It will be seen that a rather larger portion of the skin of the anterior surface of the arm was rendered completely anæsthetic in this case, than in my case of rupture of the plexus. In excision of a portion of the plexus, the communicating branch from the fourth nerve would be divided, while in rupture it is most probably spared, and consequently in Maury and Duhring's case what remained of sensibility in the skin of the upper arm must have been conferred by the intercosto-humeral and the descending branches of the cervical plexus alone.

Fig. 21.

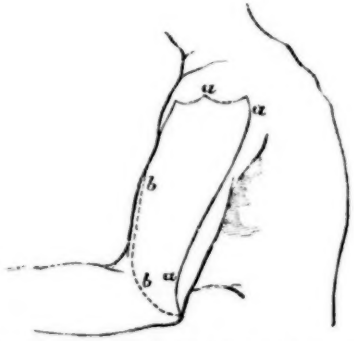


Fig. 21 (after Maury and Duhring). The line *a a a*, on the outer surface of the arm, marks the boundary above and outside of which sensation is preserved. The dotted line *b b*, marks the same for the inner surface of the arm.

The only cases which I have mentioned in this paper are those in which one or more of the cutaneous nerves of the brachial plexus had been completely divided, and in which the permanency of paralysis in the region of distribution of the corresponding motor fibres, or some other circumstance, had shown that no reunion of the divided ends could have taken place. From these cases the following conclusions may be drawn.

1. That one of the principal nerve trunks of the brachial plexus may be completely divided without giving rise to

complete anæsthesia in any part of the area of distribution of the sensory branches of the nerve; and that when complete anæsthesia does occur the portion of skin affected is very limited, and even the area of skin affected with partial anæsthesia is usually much less than that of the district corresponding to the anatomical distribution of the nerve.

2. That, as a general rule, the anæsthesia caused by division of one or more cutaneous nerves tends to become progressively less in degree and extent with the lapse of time. From this it follows that in division of one or more cutaneous nerves the area of normal sensibility tends to encroach upon the anæsthetic district, so that when one nerve (say the radial) is divided, its area of distribution, when judged by the extent of the anæsthesia, appears to be very small, as in Fig. 16, while when it is the only one of the three principal nerves of the hand that is spared, its area of distribution, as judged by the extent of the area of normal sensibility, appears to be unusually large, as in Fig. 9.

3. That the extent, degree, and even localisation of the anæsthesia caused by division of any nerve differ greatly in different cases, without our being able to discover anything in the nature of the injury to the nerve or in the external circumstances of the patient which would account for these differences (Figs. 15 and 16).

4. That the descriptions given by Krause and by Henle of the anatomical distribution of the digital branches, and especially of those derived from the median nerve, correspond more accurately with the results of pathological observation than those usually given in English anatomical works.

ON SOME PECULIARITIES CONNECTED WITH RETINAL IMAGES.

BY SYDNEY HODGES,

Associate of the Royal Academy.

In the 'Nineteenth Century' for October last, I detailed at considerable length some experiments I had been making in connection with the phenomena of after-images. It is not desirable to repeat my description of the experiments here. Those who are interested in the subject will have no difficulty in referring to them in the magazine in question. I wish, however, to recapitulate a few main points, by way of introduction to some physiological facts which will probably prove of more immediate interest to the readers of 'BRAIN.'

The main conclusions at which I arrived, after devoting several months to careful experiments, are the following:

1. That the colour of an after-image produced by gazing steadily at bright light or a strongly illuminated surface, such as white paper in sunlight, is not in any way dependent on the colour of the object we look at, but upon the amount of light thrown on the retina; either by the greater or lesser intensity of light in the object itself, or by the amount of time during which one looks at it.

2. That the succession of colours the image undergoes in forming and fading follows the order of the colours of the solar spectrum.

3. That the image is intermittent if the exciting object is looked at with one eye only, and intermittent in colour if it is looked at for a certain interval with one eye, and then for a similar interval with both.

These, briefly stated, are the main results of my observations. With reference to the first point, I may add that the best after-images are produced by placing a half-sheet of white note-

paper in sunlight, and then fixing the gaze with absolute steadiness on a small pencil-mark in the centre. If the sun is shining brightly, and I look at a paper thus exposed for ten seconds and then close and cover the eyes, I get a blue after-image. If I look for fifteen seconds, I get a green image. If I look for twenty-five seconds, I get a yellow image; but no amount of exposure ever produces any colour beyond the yellow. With reference to the second point, if the eyes are kept closed, the colour of the image changes first to dull orange, then to pink, then to crimson, then to violet, and, after an interval, back to blue; thus, as I have before observed, following the order of the colours of the solar spectrum. After the image returns to blue, it darkens and fades altogether.

With regard to the third point, namely the intermission of the image if looked at with one eye only—which fact seems to be considered the most valuable discovery from a physiological and probably from a pathological point of view—I would remark that various experiments have led me to the conclusion that, *under certain circumstances*, one eye, if rigidly fixed, is incapable of a sustained impression of vision. The reasons among others, which have induced this belief are the following:

If I fix one eye on a certain point in the sky—a small white immovable cloud is the best object—and then close and cover the other eye with the hand, in about half a minute or a minute, if the eye is kept very steadily fixed, a dark cloud like London smoke comes over the point at which I am gazing, and entirely blots it out. After an interval of a few seconds the cloud reappears, and again, after an interval, disappears.

If I look at a sheet of paper in sunlight with one eye for some twenty seconds and then close and cover the eye, I get a coloured after-image which comes and goes at intervals of six or seven seconds. This image, I should remark, is not nearly so vivid as the images formed by looking with both eyes, and it is much longer in forming.

If I look at a white object through a glass of a certain colour with one eye and through a glass of another colour with the other eye at the same time, the colours do not mingle, but the object appears first of one colour and then of the other,

alternating at intervals of a few seconds. This last experiment has been dealt with at some length by Helmholtz, but the former ones I have never seen recorded.

It appears to me that the explanation of the intermission lies in the close sympathy which exists between the two eyes, from the peculiar manner in which the optic nerves unite and again divide. Each must necessarily partake, to some extent, of the sensation conveyed to the other. For instance, after gazing at the white cloud for a certain time and receiving the impression of light in one eye, the darkness of the closed eye begins to assert itself, and insists on conveying the sensation of darkness to the sensorium before the eye exposed to light is again allowed to convey its impulse of light.

Since the publication of the article to which I have referred, I have discovered a few new facts in connection with the sensation of colour which seem to me of considerable interest. I must, however, admit that one of these is to some extent antagonistic to some theories I before advanced. In connection with the resources of the retina, I ventured to raise some objection to the "fatigue theory," so popular with Helmholtz and other physiologists. My idea was that the phenomena connected with this theory were due rather to a principle of compensation. The fatigue theory is founded chiefly on the fact, that if we look at a particular colour for a certain interval, and then remove the eyes to some plain surface, we see the complementary colour. I urged that this could hardly arise from fatigue, because if we continued looking at the original colour, although it might become less vivid, it never wholly disappeared. It appeared to me that, if it arose from actual fatigue of the rods and cones, which seem to be the seat of the primary sensation of vision, we should after a time fail to appreciate the colour altogether. This I now find, under certain circumstances, is actually the case. The simple experiment which led to my discovering this is, I think, new, and may excite some interest. It is of course known that if the three primary colours, or all the colours of the solar spectrum are painted on a disk, and the disk is made to revolve rapidly, the colours disappear and the disk assumes a greyish-white tint. I was anxious to ascertain if the sensation

of white could also be obtained by prolonged exposure of the retina to the three primaries without the revolving process. I therefore coloured a piece of cardboard with faint tints of the three primaries accepted by artists, namely red, blue, and yellow, painting these colours in stripes of equal width. I then held the card in the sunlight, fixing my eyes upon a pencil mark in the centre. In a few seconds the colours began to grow fainter, then to merge into each other, then to disappear altogether, and it appeared to me as if I were holding a piece of white card in my hand. If, however, I moved the eyes to ever so small an extent, either to the right or left, so as to bring the different tints on to another part of the retina, they immediately reappeared, and were apparently intensified in tone. This experiment seemed to me a conclusive proof that if the three rays from the three primaries were conveyed to the retina simultaneously, the sensation of white was produced, thus, apparently, at the same time proving that red, blue and yellow were the primary constituents of white light.

I next proceeded to experiment with the three colours accepted by Dr. Thomas Young, Professor Helmholtz, and other physiologists, as the primaries, namely red, green, and violet; painting faint tints of each on a card in the same manner as before. Somewhat to my surprise the result was precisely the same. When held in sunlight, the colours disappeared if the eyes were immovably fixed on them. This led me to think that it must be something apart from the combination of particular rays which produced the sensation of white. I therefore tried the experiment with two colours. Again the result was the same. I then isolated the centre colours of the first card, by covering the other two colours with white cards. The result was still the same. The colour disappeared, and the three cards presented an almost uniform surface of white. I was thus forced to the conclusion, that it was not the combination of certain coloured rays which produced the sensation of white, but that the retina was incapable of sustaining the impression of strongly illuminated faint colours on the same portion of its surface for any length of time. As I have before remarked, if the gaze was shifted to ever so slight an extent, the colour at once reappeared; which

fact leads me to the conclusion that it is only by the rapid and constant shifting of the eye that we retain the full sensation of colour. There have been so few recorded observations of the results obtained by fixing the eyes immovably on strongly illuminated objects, that this class of experiments seems to me to open quite a new field of investigation. The disappearance of the colours came upon me as a new and most surprising fact, and certainly does favour the theory of fatigue.[1] I should mention that the same effect may be produced, to a certain extent, by strong gaslight or ordinary daylight, but the result is not nearly so satisfactory.

I am anxious to record another very curious fact in connection with the intermission of the image before referred to. I happened to be looking at the sun through my telescope one morning, for the purpose of observing some sun-spots. On withdrawing my eye from the telescope, and turning towards a dark corner of the room, I found that I had a pink after-image of the sun. Remembering that it was the result of looking with one eye only, I anticipated the intermission, and found that it almost immediately commenced. I then took a rapid glance at the sun with both eyes, and then closed and covered them. The result was very curious. The intermittent after-image first produced was still coming and going at intervals of a few seconds, and through this intermittent image was seen the non-intermittent after-image produced by the momentary glance with both eyes. It should be borne in mind that the first image, having been obtained through the telescope, was magnified some eighty diameters, so that the second image was only seen as a small spot in its midst, but still it was seen in the *same field*, and therefore presents a most curious illustration of the extraordinary and complex powers and capabilities of the retina—the power to retain two distinct impressions of the same object upon the same spot.

Dr. Crichton-Browne seems to be under the impression, that in my former paper I did not make sufficient allowance for the personal factor. He considered that I possessed a special

[1] Since this was written, Dr. Tuke has sent me a paper by an American writer which induces me to think that these effects are produced by changes in the visual purple.

faculty for the production of after-images. This idea, however, is not borne out in my experiments with others. When the conditions are the same, the results have been almost identical, even with those who have experimented under my directions for the first time. Almost all the experiments, described both here and elsewhere, have been tried with others, and generally with the same results. I have no doubt, however, that the faculty of observing after-images may be greatly increased by cultivation. It is evident that the retina is never wholly at rest. Even in the middle of the darkest nights, in bed, with the eyes closed, one is conscious of certain colours passing in waves across the retina, and of flashes and rings of light resulting from sudden movements of the head. One very curious and very constant sensation is that of certain concentric rings, or waves of colour, which appear to flow from the outer circumference of the retina towards a variable disk of colours in the centre, generally of a crimson or purple tint. I have no reason, however, to suppose that even this sensation is abnormal.

NOTE ON MENTAL SUGGESTION.

BY PROF. CHARLES RICHET, M.D. (PARIS).

AMONG the various phenomena which have recently excited the curiosity of investigators, none are more interesting than those to which the name of "mental suggestion" has been given. It is true that these facts—supposing that they be facts—have long been affirmed by professional magnetisers; but none has hitherto been able to give a formal demonstration of it. The more indeed they sought to prove mental suggestion, the more it seemed to escape every scientific control.

Let us first define the term "mental suggestion."

Suggestion implies the inspiration of a thought to a hypnotised, magnetised, or normal subject. Whether this suggestion be the consequence of a gesture, word, or any other indication, it is a *material*, not a *mental* suggestion, as it were, easy of explanation, and not offering any cause for surprise. But what would be most important to prove is that this suggestion may be exercised, without the least external gesture, by the effort of thought alone. Such a suggestion, occurring independently of the least external indication, would be *mental*.

The following example will explain the difference between expressed and mental suggestion: Assuming the subject to be hypnotised and sensitive, it will be enough to describe a few zigzags with the finger on the floor, in order that the magnetised individual shall believe he sees a serpent. This is an example of expressed suggestion, and the production of the image *serpent* in the mind of the subject presents nothing wonderful, in presence of the cerebral excitation.

What would be highly astonishing is that the same image should arise without an external gesture or indication, and that the intellectual effort should, without any external manifesta-

tion, provoke the idea *serpent* in another mind. This is what many magnetisers say does take place, and it must be granted that some are of good faith, and that a few are even pretty good observers. But hitherto I am not aware that they have been able to prove the fact.

Hence he who should give a formal and scientific demonstration of mental suggestion would certainly make a true discovery, and one of the most important of our times. For the honour of a discovery does not belong so much to him who affirms a fact without demonstrating it, as to him who proves it after it has been affirmed by others. I have long sought, and am still seeking, to obtain a decisive proof of mental suggestion; unfortunately I must confess that my results have not been satisfactory, though I have had to do with some very sensitive and well-trained subjects.

My only object here is to indicate how the experiment may be performed. It is necessary, before all, that the action to be suggested mentally be simple, and not ridiculously complicated, as when the object is to deceive an ignorant public. The simplest way is to make the subject count aloud, and at any moment to give him a mental order to stop, after he has been warned that he is going to be stopped during the process of counting. In order to effect this, one must have sufficient self-command to restrain the least movement, or even the least change in the respiratory rhythm—in other words, to secure the purely mental nature of the suggestion. For the hyperæsthesia of the subject is sometimes such that the least gesture is perceived through the disturbance of the air it produces.

Following such a method, I have repeatedly ascertained that it is almost impossible to obtain a proof of mental suggestion—at any rate I have been unable to do so. It happens that the subject stops at the desired number, once or twice or three times, at the beginning of the experiment; but even this is not regular. At any rate, after the second answer the results become so divergent as to exclude every scientific deduction.

It has also seemed to me that there is a certain delay between the number mentally selected and the number at

which the subject stopped. If, for instance, the number 8 is thought, the subject, counting aloud, stops at 11.

These data, however imperfect they are, and far from establishing the almost miraculous fact of mental suggestion, will perhaps serve to some more successful experimenter for giving a demonstration which hitherto has not been obtained.

A NEW HISTOLOGICAL METHOD FOR THE STUDY OF NERVE-TRACTS IN THE BRAIN AND SPINAL CHORD.

BY DR. SIGM. FREUD,

Assistant Physician to the Vienna General Hospital.

In the course of my studies on the structure and development of the medulla oblongata I succeeded in working out the following method, which will be found a powerful aid in tracing the course of fibres in the central nervous system of the adult and the embryo.

Pieces of the organ are hardened in bichromate of potash, or in *Erlicki's* fluid (2½ parts of bichromate of potash and ½ of sulphate of copper to 100 parts of water), and the process of hardening is finished by placing the specimen in alcohol; thin sections are cut by means of a microtome and washed in distilled water. The washed sections are brought into an aqueous solution of chloride of gold (1 to 100) to which is added half or an equal volume of strong alcohol. This mixture is to be preferred to the simple aqueous solution of chloride of gold, which has been hitherto used in staining preparations; the sections are to remain in it from 3 to 5 hours. With the aid of a wooden rod (metal to be avoided) they are taken out of this solution, washed in distilled water and placed in a concentrated solution of caustic soda (1 to 5 or 6 of water); which very soon renders them transparent and slippery. After 2 or 3 minutes the preparations are taken out of the soda with the same wooden rod (toothpick or match), and the superfluous soda is allowed to drop off. The sections are then, with the soda they still contain, put into a 10 per cent. solution of iodide of potash where they almost immediately receive a tender rose-colouring, which changes into darker hues of red during the next 5 to 15

minutes. Now if the preparation be from the nervous system of the adult, it may be simply washed in water and transferred into alcohol, to be mounted in the usual way. But if it be of the brain or spinal cord of the new-born or the embryo, this treatment would spoil the section, by causing it to shrink, and throwing it into folds. Therefore these preparations must be brought upon a glass-slide by means of a camel's-hair brush, and dried by gently (without pressure) covering it with a piece of filter-paper. If the sections be very thin and soft, even this must be avoided; for the fibres of the paper would leave traces on the surface of the sections, and render them unfit for the study of the nervous elements. Nothing else can be done but to apply a piece of filter-paper to the edges of the stained preparation lying on the slide, and to draw off the alkaline fluid in this way. This is by far the most tedious stage of the process, yet it is always possible to avoid shrinking and to preserve the most sensitive preparations. The sections nearly dried are allowed to remain in water a few minutes, and are then treated in the way mentioned above.

This method will never fail (as all methods of staining by chloride of gold will do) if the specimen be not overhardened and brittle, as it is sure to be if kept in the hardening fluid an inordinate length of time. In order to stain the central nervous system of the adult, the method just described may be modified in many ways; but satisfactory preparations from the new-born or the embryo will require scrupulous attention to the details mentioned.

By this method the fibres are made to show in a pink, deep purple, blue or even black colour, and are brought distinctly into view, while the grey substance, vessels and neuroglia, lost in the slightly tinged background, are not obtruded upon the attention of the observer. A good many fine fibres, which cannot be revealed by carmine, and were not known until the methods of *Exner* and *Weigert* came into use, are seen scattered everywhere throughout the white and grey substance. In the adult the big nerve-cells also appear, and the ensemble of fibres is much too complicated for analysis; in the new-born and the embryo, the nerve-fibres alone are strikingly brought out, and those bundles, which are already possessed of a

medullary sheath, are distinguished by darker colouring from the others. Examined under higher powers, the single axis-cylinders are so well defined as to enable one to count their number.

While this method is not adapted to the study of the grey substance, it is believed that it will prove of great service in the study of nerve-tracts, particularly if the central nervous system of the new-born or the embryo be made the subject of investigation.

Clinical Cases.

DESTRUCTION OF OCCIPITAL LOBE ACCOMPANIED BY BLINDNESS.

BY D. J. HAMILTON, M.B., F.R.S.E.,

Professor of Pathological Anatomy (Sir Erasmus Wilson Chair), Aberdeen University.

As lesions involving the occipital lobe, and that only, are somewhat rare, the present case, which is almost unrivalled for the isolated character of the lesion and the complete destruction it produced, may perhaps be of interest and value as tending to locate the function of the occipital region. The patient was under the care of Dr. James, in the Edinburgh Royal Infirmary. I am indebted to him for the notes of the history. The examination of the nerve-centres was made by the method recommended by me in this journal for July 1883, and I now possess a complete series of permanent preparations of entire horizontal sections of the brain, showing the points to which I refer in the following record. The lesion and its effects could be traced out almost diagrammatically, and the method of preparation presents this great advantage, that the parts can be retained for future reference and comparison.

The patient was a female, C. R., aged 36, a mill-worker by occupation. She was admitted to the Edinburgh Royal Infirmary on August 9th, 1882, and remained there until the time of her death. She was nearly six weeks under observation in the Institution.

History.—Patient's father died about six weeks ago of a fit, the nature of which she cannot recollect. Her mother is alive, but is not strong. She has always complained of her breathing. Patient has four brothers and two sisters, all of whom are quite healthy. She has always been able to take her food well, and does not drink at all. She leaves the house only when she is going to or coming from her work. She has never had any previous illness nor accident.

About six months ago she had great sickness and vomiting.

This seems to have been connected with the taking of food, and occurred at no special time of the day. She had no particular pain during vomiting. The vomited matter seems to have been chiefly composed of what she had been eating. It was neither frothy nor of a greenish tint. She went to a medical man in town, and he prescribed iron for her. This sickness lasted for about a month. About this time she also complained of having severe headaches. After this she was subject to what she says were fits. These did not occur every day; but she had occasionally more than one in one day. She was not in the habit of crying out, nor did she bite her tongue. She had no convulsions. During them she seems to have been quite unconscious of all her surroundings, and power of vision disappeared during them. They seem to have been more of the nature of faints. They have gradually gone away, and she has not had one for more than six weeks. All this time she has complained much of headache. She has always taken the medicine she got from the medical man. When these fits were gone, she complained of her eyesight. It has gradually been getting worse and worse, till now she is almost quite blind. There is an obscure history of a squint. She cannot say how long she has had it, but it has been mentioned to her several times by different people. She cannot say if it was constantly present, or merely coming on for a short time and then passing off again. When she was first taken ill the pain was in every part of her head, but now it is confined to the front of the head between her temples. During all her life she has been troubled with palpitation.

State on admission.—Patient is not very well developed. Her muscles are small, but firm. Her grasp of the hand is feeble. She lies quite motionless in bed with her legs usually drawn up. She has a very staring look. The pupils are widely dilated. There is a slight squint outwards of the right eye. She usually lies on her right side. There is no evidence of disease or previous injury on her person. She has an extremely anxious expression of face.

Circulatory System.—She has no pain in the region of her heart, but is much troubled with palpitation. When she walks quickly, or has anything to annoy her, she is troubled with shortness of breath. The præcordia is natural as regards form and shape. A strong impulse is visible between the 5th and 6th ribs in the mammillary line, but it is rather diffused. The impulse on being felt is of a powerful and thumping character. It is apparently followed by a much slighter impulse.

On percussion, the deep dulness is as follows:—The upper border is on a level with the 2nd left rib, the right border is

in the level of the 3rd and 4th ribs, in the parasternal line, the apex-beat is slightly to the outer side of the mammillary line between the 5th and 6th ribs. In the mitral area, on auscultation, both sounds were found to be of natural rhythm, but are rather feeble. In the other areas the sounds are similar. The pulse at the wrist is feeble, small, easily compressible, but regular.

Respiratory System.—There are 24 respirations a minute. The breathing is quiet and regular. It is chiefly abdominal. She has a slight cough which does not trouble her much, except at night. There is no spit. The chest is fairly well formed. The costal angle is very acute. The interspaces are well marked. Owing to the great prominence of the 2nd rib there seems to be some flattening under the clavicles. The fremitus is not increased on either side, but is rather better marked on the right side under the clavicles, and is not felt on either side below the 2nd rib in front. There is considerable impairment on percussion on the left side throughout the front. On the right side there is fair resonance. At both apices (1st interspace) there is great prolongation of inspiration, and there is a pause between the two. It is more or less bronchial in character on both sides. On the right side this is much better marked than on the left. On the left side fine crepitations can be heard, and the vocal resonance is somewhat increased. In the 3rd interspace the same prolongation of expiration is noticed, but below this it is normal. Posteriorly vocal fremitus is better marked on the right than on the left side. Percussion is slightly impaired on the left side. At the left apex there is bronchial breathing with increased vocal resonance. At the right apex the character is much the same. There is an impaired percussion note in the scapular and interscapular regions to within an inch of the angles, and on the left side there is also some impairment in the upper part of the scapular region. In the interscapular region the breathing on the left side is rather feeble. On the right side it is natural. Over the left scapula expiration is prolonged and breathing is feeble. Fine crepitations are heard on forced inspiration. Over the right scapula the character is the same. At the left base the breath sounds are hardly audible at all, and the same is the case on the right side. Throughout the right axilla the breath sounds are feeble.

Nervous System.—Patient could not give an account of the origin of the blindness, except that it came on very rapidly about six months ago. There was nothing except dimness of vision at first, no diplopia or colour-blindness. On admission the patient was in a very torpid state, and did not answer clearly. She was able to distinguish light from darkness, but

answered at random as to how many fingers were held up. Dr. Argyll Robertson reports that there is very marked optic neuritis in both eyes, and that the left is the more advanced.

Hearing is fairly good. There is no complaint of noises in the head. Taste is not acute, and smell is almost absent. No difference could be detected on the two sides. As to sensation and sensibility there are no abnormalities complained of, and the sense of touch is nowhere impaired. Tickling did not cause reflex movements. Organic reflexes were normal. Tendon reflex very slight. Abdominal skin reflexes were absent. As to the mental condition there were no delusions manifested, and patient gave a fair account of her past history, answering questions apparently correctly, but not volunteering any information; nothing abnormal discovered except her extreme torpidity, which persisted during her whole stay in hospital.

The torpidity grew worse, and she gradually sank on Sept. 18th, 1882, the symptoms having been present for six months.

It should be mentioned that the temperature remained normal up till within a few days before death, and even then the highest attained was 101·2 F.

The post-mortem examination was made by me on September 18th, 1882.

The result of it was the following :—

External Appearances.—Height, 60 inches. Circumference at shoulders, 29¾ inches. The rigor-mortis was fairly marked. The whole upper surface, as well as the dependent parts, presented an anæmic appearance. The pupils were both considerably dilated, and of equal size. The fingers were slightly clubbed.

Heart.—The aortic valve was competent and the organ otherwise apparently healthy.

Lungs.—Right weighed 16 oz. At the apex there was a small cicatrix with a slight cheesy deposit. The lung otherwise was healthy. The left weighed 1 lb. 4 oz. Within the upper lobe were two cavities; one at the apex about the size of a small orange, the other, somewhat smaller, towards its base. They were not ordinary phthisical cavities, but had more the characters of gangrene. The uppermost cavity had a strong gangrenous odour. Their edges were infiltrated with some solid tissue, apparently cancerous, which also extended into the bronchial glands. There had apparently been a recent hæmorrhage into the lower cavity. The lower lobe was congested, but was otherwise healthy. Microscopically the tumour was composed of large flat cells, evidently cancerous in their nature.

Liver.—Weighed 2 lb. 11 oz. Slightly congested.

Spleen.—Weighed 4 oz. Healthy.

Kidneys.—Left weighed 4½ oz. Slightly congested. The right was in a similar condition.

Stomach.—The mucous membrane was slightly congested.

The Brain weighed 3 lbs. The skull was particularly thin. The surface of the hemispheres was specially dry, and the convolutions were flattened. Occupying the occipital lobe on the left side, and entirely confined to it, there was a tumour apparently growing from the dura mater. The dura mater was closely and inseparably adherent. The tumour, so far as could be made out from superficial examination, seemed to occupy the whole of the occipital lobe. The parietal lobe was intact. The optic nerves and tracts did not appear to have undergone marked atrophy. The dura mater was easily separated from the skull.

Eyes.—Both discs were much atrophied, and the retina for some distance round them had a yellowish tint.

The brain was carefully hardened and prepared as before mentioned. The following was the result of the detailed examination.

The tumour was about the size of a billiard-ball. It was firm in consistence, and could not easily be dislodged from the brain tissue, with which it appeared to be continuous. Microscopically it presented the features of a flat-celled epithelioma. The cells were quite characteristic, and were evidently growing, as the nuclei in all of them were particularly large and prominent. They were arranged in characteristic cell-nests in some parts, and in others they had a concentric laminated appearance round the blood-vessels. The tumour was vascular, the vessels being in many cases tortuous and distended.

Round about it there was a zone of redness, about one and a half line thick, which, on microscopic examination, proved to be caused by great numbers of distended and extremely tortuous capillary blood-vessels. The tissue in this zone was made up of disintegrated brain substance. Great numbers of compound granular corpuscles could be seen, evidently resulting from the fatty degeneration of the nerve and connective tissue elements. The tumour had not pushed the brain tissue aside, but appeared to have been incorporated with it, or at any rate, to have given rise to complete destruction of it throughout the whole occipital lobe. It involved both the grey and white matter, extending forwards with the latter for a distance of something like an inch to an inch and a quarter. On the supposition that the grey matter of the occipital lobe had been simply pushed inwards by the pressure of the growth, I made

a very careful examination of the surrounding parts for the presence of nerve cells or any remnant of the cortical substance. Nothing of the kind, however, was discoverable, and I am therefore strongly inclined to believe that the grey matter of the cortex was entirely destroyed. The amount of disintegration in the red zone above described as surrounding the tumour, entirely precluded the idea of the cortical substance having been merely inverted or pushed aside. The occipital lobe in fact, in the preparations which I possess, appears to have been quite cleanly cut out, and its place substituted by the tumour mass. It was the only tumour found throughout the central nervous system, and it seems probable that it was secondary to the gangrenous neoplasm contained in the left lung.

Such was the condition of the tumour and its immediate neighbourhood, and although it was the only tumour contained in the brain, it must not be supposed that it was the only lesion. I naturally looked at first for some trophic effect upon the nerve fibres coming from the destroyed area. I question, however, whether anything of the nature of a secondary degeneration had occurred.

It will be remembered that the fibres running back to the occipital lobe are not diffusely spread out in the parietal and occipital white matter, but are confined to a ribbon-like band which lies closely in contact with the tapetum or callosal fibres lining the posterior cornu of the lateral ventricle. The borders of this band are perfectly defined. There is no mixing up of the fibres in an indiscriminate manner with those derived from the cortex proper of the parietal lobe and the posterior extremity of the ascending parietal convolution. The callosal fibres of the tapetum no doubt run through it, and there is an intercrossing of the two sets, but the margin of those coming backwards is perfectly isolated.

Gratiolet[1] was the first to describe this band, and consequently it is known at the present day usually by the name of "Gratiolet's optic radiation." He supposed that he had traced its direct connection with the optic tract.

Now this "optic radiation" contains, in fact, all the fibres going to the occipital lobe, unless those which join the corpus callosum. The latter run across the "optic radiation" and enter the tapetum, to be by it conveyed still further forwards to the corpus callosum. The fibres composing it belong to many different sets, and undoubtedly are possessed of widely diverse functions. So far as I can make out, they are the following:—

[1] "Anatomie comparée du Système Nerveux." 'Comptes Rendus,' 1854, t. xxxix. p. 274.

1. A very large band which connects the Island of Reil with the tip of the occipital lobe. It runs directly backwards, and can be seen with the greatest ease in a section made in a horizontal plane. It is derived, in front, from the *unnamed white substance of the Island.*
2. A large band of fibres, supposed to be sensory in its function, which turns directly into the posterior third of the posterior division of the inner capsule.
3. Fibres which directly enter the optic tract of the same side.
4. A band to the corpus geniculatum internum.
5. Another to the corpus geniculatum externum.
6. A third to the pulvinar.
7. Fibres to the thalamus itself.

These all run directly back to the occipital region in a single band; and although this band does become smaller as it passes backwards, yet by far the greater part of its fibres end in the occipital cortex. In fact, the occipital lobe is made up of the expansion of this band and the fibres which enter the corpus callosum.

If, therefore, a secondary degeneration were to occur it would in all probability be located in the "optic radiation," or in the tapetum. My attention was accordingly directed to the examination of these, with the following results. Immediately on the same plane as the centre of the tumour, the "optic radiation" showed some amount of disintegration. It could be seen with the naked eye, because of the degenerated part being clearer than the surroundings. The degeneration was situated in the middle of the "optic radiation," and was noticed in only a few preparations. The greater extent of this occipital band was perfectly free from anything resembling a secondary trophic lesion. On examining the degenerated part microscopically, it was found to contain compound granular corpuscles, and the nerve fibres in it were destroyed. It ran forwards for a distance of about an inch and a half, and then seemed to terminate. In case the degenerated fibres had passed into a different plane, I examined sections above and below, but in none of them could I find any trace of the continuation of the degenerated fibres. I am strongly inclined to believe that the degeneration terminated here, at least in most of the fibres.

The question comes to be, was this destruction of the central portion of the "optic radiation" a truly trophic one, or was it a continuation forwards of the inflammatory zone round the tumour? The fibres which degenerate secondarily in lesions of the cortical grey matter are essentially those which are motor in their function. This is strongly insisted upon by Charcot,

and it is a fact which is now generally recognised, that while a destructive lesion situated in the neighbourhood of the fissure of Rolando, will almost certainly, in course of time, give rise to secondary descending degeneration in the motor fibres derived from that neighbourhood, similar lesions may be located in the prefrontal area without causing anything of this kind. Very much the same thing appears to hold good of the occipital region, and I am strongly persuaded that the greater part of the destruction of the "optic radiation," above referred to, in my case was purely inflammatory. The vessels within it were all distended, and there were other evidences of the destruction of the nerve fibres having been brought about by causes other than trophic influence. I will not say, in the absence of clear proof to the contrary, that the direct fibres entering *the optic tract* from the "optic radiation" did not so degenerate, but I think, as regards the other bundles which compose it, there was no evidence of a progressive secondary trophic destruction of their fibres.

While, however, the destruction of the occipital lobe was unaccompanied by distinct evidence of secondary degeneration in the fibres springing from it, the lesion had given rise to widespread secondary changes of quite another description. These mainly consisted in an enormous enlargement of the cerebral hemisphere on the same (left) side as the tumour, while the right hemisphere remained of natural dimensions. In the preparations which were made through a plane corresponding with the centre of the tumour, the left cerebral hemisphere was at least twice as large as the right. The enlargement was universal, the frontal region being increased in size in the same manner as the parietal. The enlargement, however, was greatest in the parietal, although the proportional difference between that of the frontal and parietal regions was not very marked. The right side of the brain did not seem to have suffered at all, with the exception of a slight congestion of the occipital lobe, and, so far as could be seen, looked quite normal. The corpus callosum was thickened on the left side, not markedly on the right, and it was curiously twisted, owing to the displacement caused by the increased bulk of the diseased hemisphere. The lateral ventricles were also much twisted, and the left was in parts almost obliterated, from the protrusion into it of the mass of the neighbouring hemisphere.

The grey matter of the diseased hemisphere did not seem to have undergone much alteration in size. It was of almost exactly the same size as that in the other. Nor did its texture appear to be altered, the increase in bulk being almost entirely confined to the white matter of the centrum ovale and fibres coming from it. The white matter of the temporo-sphenoidal

lobe had undergone the same enlargement that other parts of the corresponding hemisphere had.

The affected hemisphere presented an unnatural toughness. It was homogeneous when examined in a preparation clarified by my method, and the bundles of fibres, such as those of the inner capsule, did not seem to be at all destroyed, although they were separated by a much greater mass of intermediate tissue than is commonly met with. Those at the genu of the inner capsule were even more distinct than usual, evidently on account of their separation by intermediate tissue. As regards the basal ganglia, they appeared to have sustained very little injury. The bundles of fibres running into them were thickened, and this gave to the thalamus, at least, an appearance of enlargement. The grey matter, however, was unaltered, and did not seem to be swollen like the white. In fact, the increase in size seemed to have been exclusively confined to the white matter, any alteration in the grey being rather that of diminution in bulk from the pressure exerted upon it by the former. The thalami were considerably more congested than normally and equally so.

When the cause of this enlargement was investigated microscopically, it became evident at once that it was located in the connective tissue, not in the nerve elements. In preparations examined in Farrant's solution, a dense plexus of fibres was seen lying in the situation of the neuroglia, with here and there nuclei or glia-cells, as they might be termed, lying among them. The real character, however, of this newly-formed neuroglia was brought out only when the preparation was *half clarified* in oil of cloves, and mounted in gum damar solution. It then became manifest that the whole of this plexus of fibres and nuclei was resolvable into a dense mass of so-called "spider" or Deiters' cells. The appearance was exactly like that of a glioma I previously described in the 'Journal of Anatomy and Physiology' (vol. xiv.). The fibres were in reality enormous leashes of processes, which came off from the side of flat, many nucleated, connective-tissue corpuscles. They ran off in all directions, and, by interlacing, produced the appearance of a dense fibrous plexus. It was the growth of these that had caused the enlargement of the hemisphere.

Curiously enough, the nerve fibres had evidently not undergone degeneration to any very great extent, although they lay imbedded in this connective tissue, and surrounded by it on all sides. Strands of them could be seen running through the dense plexus of cell processes without any discontinuity or other evidence of destruction.

The optic nerves were examined with great care. In both,

the fibrous septa running in from the pia-sheath were all considerably thickened and highly nucleated. In the left, however, this condition was very much better marked than in the right, and more so in one locality than in others. The particular locality to which I refer lay near the surface of the nerve (I cannot say which part), and occupied about one-third of the circumference. The septa were all much more thickened here than elsewhere, and more densely nucleated than in other parts. The nerve bundles contained in them were decidedly altered. The normal bundles of nerve fibrils, when seen on cross section in an unclarified preparation, are almost quite opaque. In the bundles enclosed within these thickened septa they had become more transparent than usual. They had a shrunken appearance, and when highly magnified the fibrils were seen to be disintegrated and in great part destroyed. They did not, however, contain compound granular corpuscles, nor were the intermediate nuclei more abundant than usual. Minute oil globules were, however, occasionally present in them. The other fibres did not present any of these appearances, and, with the exception of the thickening of the fibrous septa, the nerve seemed to be normal. The tract of degeneration above described was found in the left nerve only; the fibres in the right nerve seemed to be quite healthy. The vessels of the optic nerves did not show any evidence of distension nor of congestion. The small arteries in the pia sheath had a thicker middle coat than usual, but there was no evidence of undue vascularity. The subdural and subarachnoid spaces of the nerve were not in any way altered.

The corpora quadrigemina, cerebellum, medulla oblongata, and cord were all carefully examined, but no very particular lesion was found in them. The central canal of the cord was nearly obliterated by desquamated epithelium.

A FATAL CASE OF CONCUSSION OF THE SPINAL CORD.

BY SEYMOUR J. SHARKEY, M.B.,

Assistant Physician and Joint Lecturer on Pathology at St. Thomas's Hospital.

E. A., æt. 25, was admitted into St. Thomas's Hospital, under the care of Mr. Francis Mason, on January 27th, 1883, and died on March 12th, 1883. I have to thank my colleague for permission to publish the following account:

The patient was one of six persons upon whom a house fell; she was found with only her head protruding from a large heap of bricks and rubbish, and was brought on a stretcher to the hospital.

On admission, she was said to have been struck upon the back-bone, and she complained of extreme pain across the sacrum and of numbness over the left buttock. There was no anæsthesia and no paraplegia, nor was there any external evidence of fracture or of other gross injury. There was, however, retention of urine.

An abnormal condition of the right leg and arm was present, but this had existed from birth. It consisted of partial paralysis and atrophy of both limbs without loss of sensation, but with some contraction of the flexor muscles of the arm. The patellar and plantar reflexes were exaggerated on this side, but there was no ankle clonus.

During the first week she was in the hospital, there was complete retention of urine and the bowels only acted once, and that after an enema. After this, incontinence of urine and fæces set in, and persisted till death.

At the same time that the evacuations began to pass involuntarily, a bedsore suddenly appeared on each buttock. They occurred first as blebs situated on the lower edges of the buttocks, one on each side of the perinæum. They were perfectly symmetrical, and, as the patient lay on her back, they were quite free from contact with the bedding. They soon burst, and then became ulcers. About a week later a swelling made its appearance on the sacrum, which was angry-looking and inflamed, and rapidly became an open bedsore.

The temperature up to the time of the incontinence of urine was only slightly raised, generally ranging at its highest

between 100° and 101° F.; but after this, up to the time of the patient's death, it was more elevated and of the hectic type, varying from normal to about 103°. This was accompanied by an ammoniacal condition of the urine and other signs of cystitis.

She died gradually of exhaustion on March 12th, and during the last weeks of life the acute bedsores began to heal.

Post-mortem examination.—Body emaciated, legs wasted, the right more so than the left; right arm also wasted and flexed at the elbow. The diminution in size of the right arm and leg appeared to affect the limbs *en masse*, and not to be due to wasting of any particular muscle. There were three bedsores, one of which was over the sacrum. The other two were remarkably and symmetrically situated. As the body lay on the table and the observer stood at the feet, two circular sores, about 1½ inch in diameter, could be seen on the buttocks, so situated one on each side of the perinæum, that they nowhere came in contact with the table. They were evidently healing, as a margin of pinkish-white, transparent, new skin, about ¼ inch broad, surrounded them.

There was no evidence of gross injury anywhere, either in the skull, vertebral column, muscles, ligaments, or long bones.

There was acute cystitis with thickening and contraction of the walls of the bladder, and likewise acute inflammation and dilatation of both ureters and thickening of their walls. The inflammatory process extended into the pelvis and calyces of the kidneys, and then in streaks through the medullary portion of the organs to reach the cortex, where the streaks ran into irregular groups of suppurating points. Both ureters contained pus, the right in the upper half of its course towards the kidney containing more than the left. This inequality seemed to be due to the presence of a slight stricture in the right ureter, halfway between the bladder and the kidney. The heart, lungs, and other viscera were normal.

On opening the spinal canal no fracture of bone, thickening of membranes, or hæmorrhage was observed; all appeared normal. In its lower half the cord was normally firm, except two or three inches in the upper lumbar region, where it was soft and slightly wrinkled transversely. The upper half, though not evidently pathologically softened, was not so firm as the lower lumbar region. In several places the right anterior cornu looked smaller than the left.

The brain was unsymmetrical, the whole of the left hemisphere being smaller than the right, and the temporo-sphenoidal lobe and angular gyrus more seriously malformed. This was evidently a congenital abnormality, and it has been fully described by me in a paper read before the Medico-Chirurgical Society on the 8th of May, 1883.

On microscopic examination of the cord sclerosis of the right lateral column was found, which was evidently in connection with the abnormality of the left cerebral hemisphere. The changes found in the softened portion of the lumbar region were distension of the perivascular spaces, and a soft, porous condition of the grey matter adjacent to the grey commissure on each side, but there were no distinct traces of inflammation.

Microscopic examination of the lumbar enlargement revealed nothing abnormal, except a vessel here and there with a few leucocytes around it.

Remarks.—With regard to the spinal cord the autopsy must be considered to have yielded negative results. For the softening in the lumbar region cannot be taken as evidence of disease, this being the position in which post-mortem softening is most frequently found; and of the latter process this cord probably afforded a specimen.

Nor did the microscope furnish any positive proofs, though it did some suggestions, that recent disease had been at work, and that the cord had all but completely recovered by the time the renal affection proved fatal.

If by concussion of the spinal cord be meant disease of the spinal cord set up directly by the shake of an accident, and not indirectly by the pressure of a fractured or dislocated bone, or of a hæmorrhage external to it, or by any such primary result of the accident, then it seems to us that the present case affords an example of concussion of the spinal cord. As no injury to bones, muscles, ligaments, &c., was detected on careful examination, we must assume that if the symptoms proceeded from disease of the spinal cord at all, the latter resulted directly from the force of the accident, whatever may have been the precise mechanism by which the result was produced.

The question then is, Was this a case of disease of the spinal cord?

It is true there was an absence of any loss of muscular power or of sensation, which are the usual phenomena produced by affections of this part of the central nervous system; but, on the other hand, other symptoms were present which are very common concomitants of paralysis and anæsthesia in acute disease of the cord, viz. (1) Retention and then incontinence of the evacuations, and (2) so-called acute bedsores.

These conditions must have been due either to direct local injury of the parts affected or to nerve lesions, either peripheral or central. We may set aside the idea of local injury of the bladder, bowel, and perinæum, as there never was the slightest evidence of it. The question only remains whether the symptoms were due to peripheral or to central nerve lesion. It is difficult to suppose that the nerves supplying the bladder and

rectum should have been directly injured, when at the post-mortem examination not the slightest damage to other parts could be discovered; and it would be still more incredible that a precisely symmetrical lesion should have affected the perineal nerves. The only reasonable explanation of the exact symmetry of the acute bedsores in the perineum, which were situated on parts not exposed to pressure, is that the cord was diseased at the spot in the lumbar region where the perineal nerves approach or meet each other. And this would equally explain the affection of the bladder and rectum.

But it may be objected that no disease of the spinal cord was actually found. To this we answer, that physiological reasoning in this case is of more value than arguments based on our inability to find pathological changes, and that for two reasons:

1. Because it is impossible to examine more than a small portion of the cord microscopically. And although such scant examination shows us the anatomical changes of disease when extensive, yet our not finding alterations in the few sections we examine is no proof of the absence of a small focus of disease. In the present instance the focus must have been very limited in extent.

2. It is legitimate to suppose, that if we had had an opportunity of examining the cord a week or so after the injury we might have found disease; and yet that six weeks after the accident all morbid changes, except the few doubtful ones recorded, might have cleared up. For the bedsores had long been healing, and death was the result not of progressive cord disease, but of an unfortunate cystitis and suppurative nephritis which early accompanied the paralysis of bladder.

It seems then that the only reasonable conclusion is, that owing to the concussion of the spinal cord produced by the force of the accident a small focus of disease resulted, which would probably have ended in the recovery of the patient, had not secondary disease of bladder and kidneys unfortunately proved fatal.

Satisfactorily proved cases of concussion of the spinal cord are undoubtedly rare; but we must remember, that in order to prove them we require a post-mortem examination. This case at least suggests what other considerations would render probable, that in severe accidents small focal lesions may result from concussion, and although they may cause very obscure and annoying symptoms, yet as they are small in extent and have not damaged vital parts they terminate in recovery, and leave us to argue as to the cause of the clinical phenomena we observe, without giving us the opportunity of proving whether our conclusions are correct.

CASE OF CONCUSSION AND INFLAMMATION OF SPINAL CORD FROM GUNSHOT-WOUND OF BACK.

BY WALTER EDMUNDS, M.D., F.R.C.S.

On October 29th, 1883, A. B., aged 19, was shot in the back; he fell immediately, and had to be carried out of action. The surgeon who first examined the case reported, "he was found to be suffering from a gunshot-wound, the projectile, apparently a large slug, having entered obliquely near the inferior angle of the right scapula; the wound was large, its edges being inverted, contused and irregular. A careful examination was made; but the finger failed to discover any foreign body, the projectile having taken a tortuous course towards the dorsal vertebræ, where it is probably lodged. There is little doubt that the spinal cord has been severely injured, for there is complete paralysis below the seat of injury."

Patient was sent home, and on coming under our observation (Nov. 25th) the wound was seen to be on the level of the fifth dorsal spine and about an inch to its right. Probe would only enter about half an inch. He was completely paraplegic, and without any sensation below the level of the wound. No control over bladder or rectum. Both arms very weak, the right weaker than the left. Some cystitis. A bedsore over sacrum. He died March 5, 1884, four and a half months after receiving the wound.

At autopsy, the bullet wound had healed; no fracture, or indication of fracture, or dislocation of vertebra could be found, nor any track of the projectile. Theca vertebralis intact. The cord was seen to be much atrophied and softened about the level of the wound. The bullet could not be found, although carefully looked for. Kidneys hypertrophied: four or five ounces of serum in pericardium.

On hardening the cord in Müller's fluid, it was seen that there was universal myelitis and softening for about two inches opposite the wound; this gradually passing below into the sclerosis of the lateral and anterior pyramidal tracts, and above into sclerosis of the posterior median columns.

There was no indication of hæmorrhage, either external to or into the substance of the cord. It surface was uninjured.

Remarks.—Although the surgeon who first examined the case was no doubt justified in his opinion, yet the subsequent progress of the case ought to have thrown some doubt on the diagnosis.

The length of time (4½ months) the patient survived must be most exceptional in fracture of vertebra with direct injury to the cord.

Lidell,[1] speaking from an experience acquired in the American Civil War, says, "Leaving out of calculation such fractures as involve the spinous processes alone, the writer has never seen a case of gunshot fracture of a vertebra get well, and he might add he has never seen life prolonged for a month after the infliction of that injury."

And further, in a fracture of bone with the bullet imbedded in it, it would be surprising that the sinus should completely close and without the discharge of spicules of any bone.

Somewhat similar cases are to be found on record. Dr. Charlton Bastian[2] relates the case of a man who fell in his sleep from the top of a haystack 25 feet high: his legs were at once paralysed, and after suffering from spinal symptoms for six months, he died. At the post-mortem examination no changes whatever were seen, for the important changes which did exist in the cord were only rendered visible by keeping it for some days in a hardening solution.

Sir William Gull records a case due to an injury to the back of the neck, terminating fatally in thirty-four hours. At the post-mortem examination no fracture or dislocation was found, but a contusion of the cord at the level of the 4th and 5th cervical vertebræ.[3]

Leyden[4] mentions a case which he saw, in which concussion of the spine causing paralysis proved fatal in five days. At the post-mortem nothing whatever could be found abnormal, either microscopically or macroscopically.

In conclusion, it may be remarked that the myelitis which was found in this case could not have accounted for the paraplegia which immediately followed the injury. The term "concussion" has been used to indicate the cause of this.

[1] Lidell, "On Injuries of the Spine, including Concussion of the Spinal Cord;" 'Amer. Journal of Med. Sciences,' 1864.
[2] 'Transactions of Med.-Chir. Society,' vol. l., 1867.
[3] Guy's Hospital Reports, 1858.
[4] 'Klinik der Rückenmarks-Krankheiten,' vol. ii., 1875.

Critical Digest.

ON THE SO-CALLED HEREDITARY ATAXIA, FIRST DESCRIBED BY FRIEDREICH.

BY J. A. ORMEROD, M.D.

REFERENCES TO PUBLISHED CASES:—

Friedreich's writings on the subject will be found in Virchow's Archiv, Bd. 26, 27, 68, 70.

Schulze gives the morbid anatomy of one of Friedreich's cases in Virchow's Archiv, Bd. 79.

Other cases are given by:—

Carré. 'De l'Ataxie locomotrice progressive.' (Thèse de Paris.)
Brousse. 'De l'Ataxie héréditaire.' (Paris, 1882.)
Kahler und Pick. 'Archiv für Psychiatrie,' &c., Bd. 8.
Seeligmüller. 'Archiv für Psychiatrie,' &c., Bd. 10.
Rutimeyer. 'Virchow's Archiv,' Bd. 91.
Schmid. 'Correspondenzblatt für Schweizer Aerzte.' 1880.
Carpenter. Medical Society of London, 1871.
Dreschfeld. Manchester and Liverpool Hospital Reports, 1876.
Gowers. 'Transactions of Clinical Society.' Vol. xiv.
D'Arcy Power. St. Bartholomew's Hospital Reports, 1882.
Kellogg. 'Archives of Electrology and Neurology.' Vol. ii.
Hammond. 'Journal of Nervous and Mental Disease," 1882.

FRIEDREICH, after describing the six cases first observed by him, gives the following general description of the disease:—

"There exists a chronic inflammatory degeneration of the spinal cord, leading to atrophy; which develops chiefly at the period of puberty, under the influence of hereditary predisposition; which is essentially limited to the posterior columns of the cord, begins in the lumbar region, spreads from thence upwards and downwards, and finds its limit in the medulla oblongata, after involving the trunks and nuclei of the hypoglossal nerve. . . .

"The clinical characters of the disease are impairment in the co-ordination and harmony of movements, developing gradually and spreading from the lower to the upper half of the body, and always involving finally the organs of speech; sensibility and the functions of the special senses, and of the brain, being intact. Paralysis of the sphincters and trophic disturbances are absent.

Less constant phenomena are curvature of the spine, sensations of vertigo, and nystagmus.

"From a clinical point of view we must regard the disease as a progressive paralysis of the faculty of the combination of movements; from the point of view of pathological anatomy, as a chronic degenerative atrophy of the posterior columns of the cord."

We shall give an abstract of two of his earlier cases, which possess the advantage of having been observed by him over a long period of time.

1. Charlotte Lotsch was one of a family of five children. Three were healthy, one (a brother) had the same disease as herself. She was well till 18, then, without assignable cause, weakness began first in the left leg, then in the right. This became worse in six years' time after a confinement; she now needed support when walking. At 26, weakness in both upper limbs appeared, and difficulty of speech. She was first seen by Friedreich at the age of 31, having been ill thirteen years. She was a spare muscular woman, yet unable to walk or stand without support. When lying down, she could execute simple movements of the legs, though with considerable effort. Simple movements of the hands and arms were executed easily, but finer movements, such as picking up a small object, threading a needle, &c., with very great difficulty. Her speech was thick and stammering. Sensibility in all its modes was normal, as was also the electro-contractility of the muscles.

A year later she had a sudden attack of diabetes insipidus, accompanied with a rise in the pulse-rate, flushings, and erythematous eruptions. These disappeared equally suddenly in three and a half months' time; transient salivation followed, also sweatings, diarrhœa and vomiting. The ataxia and the speech became worse, troublesome vertigo and palpitation set in. Before leaving the hospital (which she did in three years' time), the motor power had diminished in the lower limbs, the electro-contractility of their muscles was impaired; contracture of the feet and slight spinal curvature were noted.

She again came under observation at the age of 49, in the 31st year of the disease. In the interval she had experienced frequent cramps and pains in the lower limbs, and occasional difficulty of micturition, and she had lost power in the legs. Her state was now as follows. The lower limbs were paralysed almost completely, the muscles of the legs were flaccid, but there was frequently contracture of the thigh muscles. Sensibility was considerably diminished; the plantar reflexes were present, the patellar tendon reflexes absent. In the upper limbs the muscular power and cutaneous sensibility were unimpaired, but the incoordination had increased. There was well-marked curvature (angular and lateral) of the spine; she sat all hunched together, the head exhibiting oscillatory movements like those seen in a person going to sleep upright. The speech was almost unintelligible, nystagmus had developed; though there was no squint, and the action of the pupils was normal.

Agan there occurred a transitory elevation of the pulse-rate. She died of bronchitis, age 49, in the 33rd year of the spinal disease.

The post-mortem we shall give afterwards.

2. Friedrich Süss belonged to a family of five children; only one of them was healthy, three others were diseased like himself. At 15, he became affected with a weakness of the left leg, which in eighteen months spread to the left arm. Soon the right leg and arm became similarly affected. He had feelings of weakness in the back and loins. At 18, difficulty of speech showed itself. On rare occasions he had numb feelings in the left arm.

When first examined by Friedreich (being then 24, and having been ill nine years), he could still walk with help. He walked with a tottering uncertain gait like that of a drunkard, with peculiar tossing movements of the legs, particularly well seen on going upstairs.

He had difficulty in standing, even with open eyes, and could only do so with tottering and balancing movements of the body. Delicate movements of the hands were made with much uncertainty and irregularity. All the sensory functions were unimpaired. The speech was thick and stammering. There was slight curvature of the dorsal spine.

He was re-examined three years later. The incoordination had increased, but except that he had become subject to vertigo, and had lost power of erection and emission, there was no additional symptom. Thirteen years later he came to Friedreich again. Now he could no longer stand, owing to difficulty in balancing himself, and even when sitting, his head, if unsupported, nodded and swayed. But still there was no true paralysis, and no affection of sensation. His speech was barely intelligible. A slight lateral nystagmus, only evident on fixing, had made its appearance. He died, a year later, of typhoid, at the age of 38, the duration of the spinal disease being twenty-three years.

Let us proceed to examine the clinical features of the disease more in detail.

The more striking feature is its tendency to attack many members of one family. With regard to ordinary tabes, most authorities agree that it does not run in families, so that this constitutes a first clinical point of difference. Not that Friedreich's disease is usually hereditary, in the strict sense of the term (though it may be so), but that it attacks many members of the same generation. Table I. shows this in every instance, with the possible exception of Brousse's case, who does not mention whether the patient had any brothers or sisters. Very remarkable in this respect are Rutimeyer's observations on the Blattner family; where in the same generation, from four separate branches of the family, eight members were affected. Yet that the disease may be transmissible is shown in this same instance, for it appears probable that a male ancestor five generations back was ataxic. Again, in Brousse's case the mother of the patient was ataxic; and in Carré's (besides the brothers, sisters, and maternal cousin) the mother, maternal uncles and aunts, and grandmother. So that

successive as well as simultaneous outbreaks are possible, though not the rule. Apart from ataxy, proclivity to other forms of disease has been observed in those families; on the side of the nervous system, to chorea, paralysis, hysteria, mental affections, intemperance; on the side of general disease, to phthisis.

Friedreich's opinion, based on his own nine cases, was that females were much more prone to the disease than males, but subsequent observations show that this is not the case.

Secondly, it attacks in early life. Ordinary tabes is a disease of middle age or of later life. Doubtless we have now means of recognising it at an earlier period than formerly we could; but still its onset is less early than that of the hereditary form. Friedreich stated that the latter coincided with commencing or developing puberty, the dates in his cases varying from æt. 13 to æt. 18; but other authors have observed it to begin still earlier. Thus, in three of Rutimeyer's cases it began about the age of 4 (one child indeed had never been able to walk straight); and in two of Hammond's cases at a similarly early age. Table II. shows that for members of the same family there is usually a curious coincidence as to the dates of onset.

The first system is almost invariably ataxy of the lower limbs. This statement will hold, we believe, in spite of certain recent criticisms. Hammond, commenting on Friedreich's cases, says, "the first symptom observed was weakness of the lower extremities, and this gradually extended so as to involve the upper extremities. Here was, therefore, a true paralysis." But we must remember, that as early symptoms generally come through the report of the patient or his friends, any impediment to walking (whether paralytic or ataxic) would probably figure as weakness of the legs. If we look to the result of subsequent medical observation, we shall find many carefully observed cases in which there was ataxia but no paralysis. It is true that absolute paraplegia is recorded in some cases, and paresis of the lower limbs in others, but this was not till the disease had existed for years.

The absence of sensory symptoms is a point of great interest. In typical tabes, as is well known, the pains are usually a prominent symptom, especially in the early stages, and are rarely absent throughout. Further, sensory disturbances, such as numbness, anæsthesia, delay in the transmission of painful impressions, frequently accompany the incoordination of movement. But in the majority of hereditary cases pain is conspicuous by its absence, at least till the disease is inveterate. (There are a few exceptions, to which we shall return.) Again, though diminution of sensibility has been often noted, it is usually, for the earlier periods, in quite an insignificant degree, and in many cases every abnormality of sensation has been thoroughly excluded. We have, therefore, in this disease an illustration of ataxy pure and simple, apart from motor or sensory paralysis.

Harmony in muscular contraction is needed, says Friedreich, as much for the maintenance of a position as for movement. The lack of it during movement produces the staggering gait, the

awkwardness of the hands, &c., i.e. locomotor ataxy; the same defect in the maintenance of position he calls "static ataxy." It is illustrated in this disease by the swaying of the head and neck when unsupported, by the wavering motions sometimes seen when the arms are held out, and is a later phenomenon than is locomotor ataxy. The well-known "Romberg's symptom," i.e. inability to stand with the feet together and the eyes closed, is ascribed by Rutimeyer to static ataxy in the muscles of the trunk. This is not always present. The disease generally spreads upwards with rapidity. In many cases the arms have been attacked very soon after the legs, or at least within a year or two. But this is not invariable; four, six, eight, or more years may elapse; indeed, in one of Dreschfeld's cases the arms had not been attacked after fourteen years.

The affection of speech may manifest itself either simultaneously with the affection of the arms or not till many years afterwards. A verbal description of peculiarities of speech is more difficult even than description of gait; but this much we may say, the defect is a defect of articulation, not of word-memory; neither on the other hand is there loss of motor power in the organs of speech, though tremor of the tongue has been noticed. From a mere drawl, stammer, undue confluence, or undue separation of syllables, the affection may advance till speech becomes wholly unintelligible.

Nystagmus marks clinically the highest level to which the disease extends. It is bilateral, usually transverse, and is observed only while the patient fixes or follows an object. It is a late phenomenon, and therefore its absence need not tell against the diagnosis.

We have now enumerated the most striking features of the disease, viz. ataxy, first of the legs, then of the trunk and arms, imperfection of speech, nystagmus. But there are others of importance. First, the absence of knee-phenomenon. This has been found absent in every case examined for it (the older cases of course were not). In Friedreich's last case it was found absent within a year after the disease had declared itself, and we might expect from analogy to find it absent still earlier. In the healthy children of an ataxic family Rutimeyer has found the knee-phenomenon normal. Secondly, curvature of the spinal column. In most nervous diseases (disregarding, of course, those secondary to disease of the vertebræ) the spinal column is unaffected. Pseudo-hypertrophic muscular paralysis may be quoted as an exception. And indeed, as in that case, so in this, the cause is essentially the same, viz. the lack of efficient muscular support. A combination of lateral and angular curvature is frequent; but lateral curvature alone is noted five times as against angular curvature once. This we should expect, assuming the cause to be laxity of the muscular ligaments. Curvature of the spine may be a very early symptom; in Carpenter's second case, and in one of Friedreich's cases, it manifested itself two years before the unsteadiness of gait. Its occurrence may be sometimes due to mere coincidence, but this can scarcely be so in every case, considering how frequently it is noted.

There remain sundry other symptoms, in a certain sense accessory, and appertaining mostly to an advanced period of the disease. Such are true paralysis, atrophy, and contracture of muscles, and, on the sensory side, anæsthesia or pains. Perhaps we may add occasional visceral disturbances. Rutimeyer has noted an early form of contracture, viz. that the great toes become turned up towards the dorsum of the foot; the mother of one family had herself observed this in her children, and put it down as a "malum signum." Such symptoms probably denote that tracts of the cord other than the posterior columns are involved. Interest therefore attaches to the date of their appearance, since it may help us to understand the manner in which the disease spreads within the cord.

It is worth while to recapitulate the clinical relations borne by this disease to the ordinary form of tabes. The phenomena of ataxy beginning in the lower limbs and spreading upwards, surely, if at a variable rate, coupled with the loss of tendon reactions, constitute the common basis of the two affections. The prognosis quoad recovery appears equally hopeless in each. But they exhibit differences. For, firstly, the multiform nervous phenomena which may usher in typical tabes—such as lightning pains, anomalies of micturition, transient paralysis (especially in the oculo-motor district) loss of pupillary reflex; or more rarely optic atrophy, visceral crises, trophic disturbances in joints, bones, and elsewhere—all these are absent in the hereditary form; where uncomplicated ataxia is the initial and for a time the only symptom. Particularly remarkable is the absence of pain, and indeed, at the outset, of any sensory or motor paralysis. Secondly, in the hereditary form the spread of the affection to the arms is more rapid; and the disturbance of speech and nystagmus are peculiar to it. If with Friedreich we regard these also as ataxic, then we may say that the ataxia spreads more rapidly and at the same time spreads further in this than in the typical form. We have only to add the differences in family predisposition and in the age at onset (already sufficiently noticed) to complete the contrast.

Now seeing how marked the contrast is, it is not surprising that some authors should have denied the existence of any relationship between the two diseases. Thus Hammond maintains that Friedreich's cases are not examples of locomotor ataxy at all, but of disease in the medulla or cerebellum extending secondarily to the cord. An answer to this assertion is to be found in the morbid anatomy. For not only Friedreich's post-mortems (which cannot, we think, be shelved simply on the score of being old) but also the recent researches of Schultze and Brousse establish the fact that the disease is not cerebral, but spinal, and involves the posterior columns. The same answer must be made to the French authors who regard it as a variety of disseminated sclerosis. The affection of speech and the nystagmus indeed lend clinical support to that view, but, post-mortem, the sclerosis (at least as regards the posterior columns) has been found to be systematic, not disseminated. The views expressed by Erb appear to us better to

represent the facts. This author, while recognising fully the varieties exhibited by tabes, thinks that most cases may be grouped under one of two types, either the classical type or that described by Friedreich. This he regards as the only natural subdivision. To borrow botanical language, the natural order tabes contains two species, to which most individuals may be referred. The propriety of this classification is justified, rather than otherwise, by the occurrence of forms transitional between the two. Such forms are well illustrated by Carré's case (Table II., No. 21), in which there was well-marked heredity and affection of speech, yet the disease began with pains and numbness in the feet and legs, and diplopia was observed. Again, Dreschfeld's three cases (25–27), were it not for the fact that five members of the same family were attacked, resemble the classical type; for there were pains from the onset, and no affection of speech is mentioned. In both Carré's and Dreschfeld's cases the onset was rather late for that of the hereditary disease [æt. 20–23]. Again, early pains were noted in three of the Süss family (Nos. 3 to 5); and in Power's case (No. 34) there appear to have been vomitings at an early period. This appears to be the place to notice the observations of Seeligmüller. These relate to two brothers, whose parents were first cousins, and whose mother and maternal aunt were highly neurotic. The one brother had ataxy of the legs, nystagmus, and fibrillar twitching of the tongue, though his speech was normal. He was subject to migraine, dreamy states, and inverted sexual passions. The other brother was ataxic in the legs, had commencing nystagmus, and his speech was peculiar. He also was subject to dreaminess and forgetfulness. Friedreich refused to acknowledge these as cases of hereditary ataxy, doubtless on account of the mental symptoms, and because the knee-phenomenon was present. But they are worth mentioning while on the subject of aberrant forms, especially when we remember the connection between ordinary tabes and general paralysis of the insane.

The morbid anatomy of this disease rests upon six cases (Friedreich, Schultze, Brousse), in which the symptoms were well-marked during life, and careful examinations were made post-mortem, and upon one other case (Kahler and Pick) in which important anatomical data were obtained, though the clinical history was somewhat imperfect. Of these we shall give an abstract, as some parts of the pathology are still matter of discussion.

I. Andreas Lotsch (Table I., Case 1) died from typhoid when the ataxy was of twenty-one years' standing. Besides the ataxy and the affection of speech, there had been true paralysis of the adductors of the thighs, with some general feebleness of movement.

Post-mortem, the following lesions were found by Friedreich. Beneath the *dura mater*, in the lumbar regions of the cord only, there was fluid, and at a corresponding level some softening of the cord.

The *pia mater* upon the posterior aspect of the cord was milky

and thickened in its whole length, and there were soft filamentous adhesions between it and the dura. The *posterior columns* were grey, translucent, and firm (this change being most marked, and appearing to have commenced, just above the lumbar swelling, and terminating just above the bifurcation of the posterior columns at the calamus); the rest of the interior of the cord was normal. Microscopically, there was found atrophy of the posterior nerve-roots, with slight changes in the nerve-trunks of the sciatics and hypoglossals.

The microscopic changes in the interior of this, and of all the other cords, appear to have shown the ordinary characters of sclerosis; there was an overgrowth of fine fibrillar tissue at the expense of the nerve-fibres, which were found in various stages of degeneration. The so-called corpora amylacea were found in abundance, especially in those parts where the sclerosis appeared to be commencing.

II. Justinie Süss (Table II., Case 3) died of typhoid in the fifteenth or sixteenth year of her disease. During life, in addition to the cardinal symptoms of ataxia, difficulty of speech and nystagmus, there had been noted curvature of the spine, occasional pains, cramps in the lower limbs, some tactile anæsthesia of the lower limbs and some general feebleness of movement.

The post-mortem (Friedreich) gave the following results. There were changes in the *dura and pia mater* closely resembling those in the last case, together with pigmentation of the cervical portion of the pia. The cord was flattened and somewhat softened, rather above the lumbar swelling. The *posterior columns* were sclerosed from the lumbar to the cervical swelling throughout their whole sectional area, above the cervical swelling the sclerosis affected only a thin zone next their periphery. In addition to this, the *lateral columns* were sclerosed, between the lumbar and cervical swellings, but to a less degree than the posterior columns, and only where just adjacent to them. There was, as in the last case, a slight extension of the posterior sclerosis to the medulla, and also atrophy of the posterior nerve-roots.

A further examination of this cord, made after many years by Schulze, showed that the grey matter also was sclerosed, chiefly in the dorsal region, and the central canal was stuffed with small round cells. The cells of Clarke's columns were diminished in number.

III. Salome Süss (Table II., Case 4) died of typhoid when her ataxia was of twelve or fourteen years standing. Here also during life there had been feebleness of movement, and at an early period shooting-pains, cramps, and spinal curvature.

Post-mortem, Friedreich found *meningeal changes* like those in the last two cases; also thickening of the ependyma of the 4th ventricle, and in the cord the following lesions; sclerosis of the *posterior columns* in their whole length, highly advanced in the part between the cervical and lumbar swellings, in which part the posterior fissure was obliterated, the surface of the posterior columns sunken, and the cord generally flattened. Microscopically,

the *lateral columns* were found to be affected in the part where they abutted on the posterior columns. There was some atrophy of the posterior nerve-roots.

There were interesting changes in the grey matter, as follows: reaching from the lumbar swelling as far as halfway to the cervical swelling, were two canals with fluid contents, longitudinal in direction, and symmetrically placed one at each junction of the posterior and anterior cornua of grey matter, mostly within the grey matter, but just trenching on the lateral columns. These were prolonged upwards to the cervical region as foci of jelly-like material. There were less definite appearances of the same kind in the lumbar region.

Basing his remarks on these three post-mortems, Friedreich maintains[1] that the essential lesion consists in a chronic inflammation of the posterior columns of the cord, beginning in the lumbar region and extending upwards and downwards. But the affection of the posterior columns he was inclined to regard as secondary to the meningitis which he had found in all three cases.

The latter opinion he discarded in view of his next case.

IV. Friedrich Süss (Table II., Case 6) died of typhoid, having been ataxic some twenty or thirty years. There had been in his case no accessory symptoms, if we except vertigo and loss of sexual power.

The sac of the dura mater contained a good deal of fluid; the arachnoid was thickened and milky; the pia was thickened, and in the cervical region pigmented. The whole cord was thinner than normal, but the posterior columns specially small. An elaborate description of the minute changes, with plates, is given by Schultze.[2]

The *posterior columns* were sclerosed throughout (in the cervical regions the columns of Goll were the most deeply affected); this lesion extended upwards to the level of the calamus.

The posterior parts of the *lateral columns* were sclerosed in the cervical, dorsal, and lumbar regions (the intensity of the process, to judge from the plates, diminishing from above downwards). There was a thin zone of less degenerated tissue between the sclerosed portions of the posterior and lateral columns.

Except in the lumbar region, the affection of the postero-lateral columns reached quite to the periphery of the cord (sclerosis of direct cerebellar tract?); and in the cervical region there was a zone of degeneration extending round the periphery of the lateral and anterior columns to the anterior fissure.

Further the *R. anterior column* in the neighbourhood of the fissure was deeply sclerosed; this lesion reached upwards a little above the decussation of the pyramids, and had disappeared in the dorsal region of the cord (sclerosis of R. direct pyramidal tract?).

The grey matter of the cord was somewhat diminished in volume; the cells of Clarke's columns were remarkably atrophied.

[1] Virchow's Archiv, Bd. 26, pp. 453, 454.
[2] Virchow's Archiv, vol. lxx.

The posterior nerve-roots were thinned; the peripheral nerves and spinal ganglia were normal.

The medulla oblongata was small; it showed no distinct sclerosis, but there was slight increase of connective tissue and numerous corpora amylacea in the restiform bodies.

These changes are read by Friedreich as follows: assuming an insufficient development of the cord and medulla (as evidenced by their size), upon this became grafted the posterior sclerosis, which he considered in this instance too intense to be secondary to the meningitis. Upon the posterior sclerosis followed the meningeal changes, which were the medium through which the disease was conveyed to other tracts of the cord than the posterior columns. But this explanation of the spread of the disease is contested by later writers, notably by Kahler and Pick, whose case we give next.

V. Case recorded by Kahler and Pick, in the 'Archiv für Psychiatrie,' &c., Bd. viii. p. 251.

This was a woman, age 23, whose sister was said to be affected in a similar manner. Her illness began when about 16 years old, with painful swellings of the ankles, then progressive weakness of the legs. In six years' time the arms became weak and the speech was affected. When she came under observation the muscular weakness was so preponderant that it was impossible to say whether there was ataxia or not. The tendon reflexes, however, were absent. There were no pains and no contractures. The speech was slow, the syllables separated and brought out with effort. She died of phthisis. The authors found, post-mortem, some adhesion of the pia and dura mater in the cervical and dorsal regions, but quite insufficient to account for the changes within the cord. The cord was smaller than normal, and the posterior nerve-roots thinned.

The *posterior columns* were degenerated throughout the whole length of the cord, except a thin zone next to the grey matter. The *postero-lateral* columns were also sclerosed right up to the periphery (crossed pyramidal tract and direct cerebellar tract), but here also a zone next the grey matter was spared. The *R. anterior column* in the neighbourhood of the fissure (R. direct pyramidal tract) was degenerated down to the dorsal region; the L. was not degenerated, but to make up for this, the degeneration in the crossed pyramidal tract on the R. side (of the cord) extended over a larger area than on the L. side. [This the authors point out would be the result, supposing there were (as is known to be sometimes the case) complete decussation at the pyramids of the L. pyramidal tract, with partial decussation of the R.] The columns of Clarke were atrophied.

The gist of this case, which the authors assimilate to those of Friedreich, though the clinical data are confessedly scanty, is to show that the sclerosis does not spread from one tract of the cord to another by simple contiguity (since zones of healthy tissue intervened), nor yet, as Friedreich supposed, by the medium of a meningitis; but that there is a distinct and independent degenera-

tion of various tracts—of the posterior columns proceeding upwards, of the pyramidal tracts (crossed and uncrossed) proceeding downwards, of the direct cerebellar tracts, and with them of the columns of Clarke. They assert, therefore, that Friedreich's disease is not a simple posterior sclerosis, but a "combined systematic degeneration of the cord."

To this view it would appear Schultze gives his adherence,[1] though *à propos* of the case which follows, he criticises it adversely.[2]

VI. Charlotte Lotsch (Table II., Case 2) died finally of nephritis, having been ataxic for more than thirty years. In the later stages there had been paralysis of the lower limbs, with contracture of some muscles and atrophy of others; also cramps and pains.

Post-mortem, Schultze found *meningeal changes* much resembling those already described. The cord was somewhat small. There was sclerosis of the *posterior columns*, and of the posterior parts of the *lateral columns*. As in Case 4 (Fr. Süss), the periphery of the lateral columns was diseased, but the zone of degeneration did not reach so far forwards.

The *anterior column of each side* was sclerosed from the decussation of the pyramids downwards, the degenerated tract taking the form of a comma directed from behind forwards. The columns of Clarke and the posterior nerve-roots were degenerated.

The sacro-lumbales muscles were atrophied.

VII. The last case is that published by Brousse (Table II., No. 22).

This patient, a woman æt. 24, who was the subject of phthisis, died of subarachnoid (cerebral) hæmorrhage, having been ataxic for eight years.

The cerebrum was in other respects normal, the cerebellum also. The pons varolii was small. There was in this case no meningitis.

Minute examination of the cord showed the lesions to be most intense at the lumbar swelling. These lesions were as follows:—

The *posterior columns* were sclerosed in the whole length of the cord; at the lumbar swelling this sclerosis extended to the periphery of the cord; in the cervical region it did not quite reach the periphery; it was most intense in the neighbourhood of the central canal and along the edge of the posterior cornua.

The *lateral columns* were sclerosed chiefly at the junction of the anterior and posterior cornua, the lesion reached the periphery of the cord in the lumbar region only.

The *anterior columns* were sclerosed principally in the neighbourhood of the anterior cornua; in the cervical region the lesion extended to the periphery of the cord (on the L. side only).

The sclerosis of the lateral and anterior columns was well marked in the cervical region, less marked in the dorsal, and most extensive in the lumbar, where the whole area of these columns was affected.

Further, there was well-marked inflammation of the *central canal*,

[1] Neurologische Centralblatt, vol. ii. p. 290. Report of a speech by him.
[2] Virchow's Archiv, Bd. 79, p. 136.

which was full of small deeply-staining cells. Upon this Brousse lays stress, as he considers that by this means the disease spread from the posterior columns to the other parts of the cord.

In the medulla oblongata there was a patch of sclerosis occupying the posterior pyramids and reaching to the level of the hypoglossal nucleus.

Brousse holds that the sclerosis of the posterior columns is primitive and systematic; while the sclerosis in other parts is secondary to it, and is diffuse, i.e. not limited to, nor propagated along, any particular tracts of fibres.

We believe that the above abstract represents with tolerable accuracy the facts already ascertained about the morbid anatomy of this disease; we will conclude by recapitulating them.

In several cases the cord and the medulla were small in size; and this is certainly a fact of interest when we consider the hereditary character of the disease. For a cord congenitally imperfect may be the more prone to degeneration, and to degenerate at an early age.

Next from the clinical history, as well as from the fact that advanced posterior spinal sclerosis was found in every post-mortem, we may assert that this at least is an essential lesion. The changes in the medulla are slight and apparently secondary to the spinal; so in Friedreich's opinion were those which he found in the nerve-trunks; while in the cerebrum and cerebellum no trace of chronic disease has been found. We therefore cannot adopt Hammond's suggestion that the lesion is primarily situated in the medulla or cerebellum.

Thirdly, since the sclerosis of the posterior columns involves their whole length, and appears to spread upwards much as in ordinary tabes, this we must class as a systematic tract lesion and not as part of a disseminated sclerosis.

But the relation of the disease in other parts of the cord to the posterior sclerosis is as yet obscure. Does such disease of other parts always exist? if so, has it spread by simple contiguity, or by a meningitis, or by the medium of an inflammation attacking the central canal? Or have we here an instance of systematic posterior sclerosis combined with disseminated sclerosis of other parts; or, on the other hand, of systematic sclerosis of many spinal tracts at the same time?

To answer these questions we need further examinations, and especially examinations at early stages of the disease.

TABLE I.—To show the Genealogical Relations of most of the Published Cases.[1]

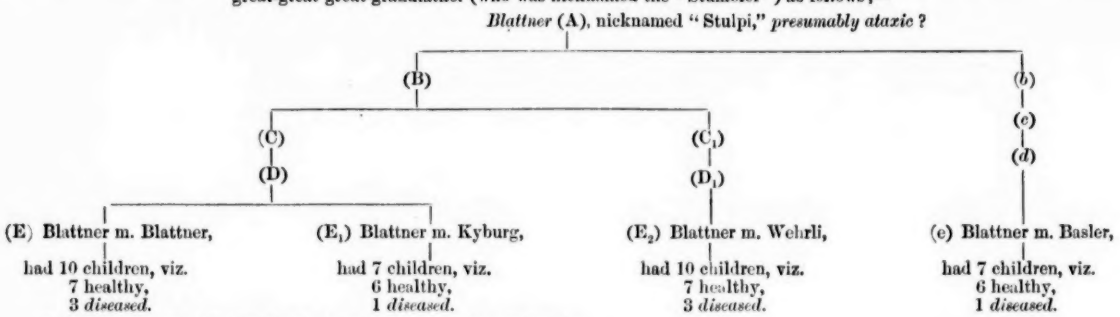
Friedreich. 1. Lotsch Family:— Father intemperate, died of dropsy. Mother died of sudden hemiplegia.	There were	5 children, of whom 2, viz. one male and one female, *were diseased.* Table II., 1 and 2. 3 were healthy.
2. Süss Family:— Father intemperate, died of phthisis. Mother living, healthy, but of a low grade of intelligence.	There were	5 children, of whom 4 *were diseased*, one male and three females. Table II., 3 to 6. 1 was healthy.
3. Schulz Family:— Father and mother living, healthy country people. No history of disease in ancestry.	There were	11 children, of whom 5 died young. 3 were healthy. 3 (all females) *were diseased.* Table II., 7 to 9.
4. Brousse's Case:— Father's mother subject to hæmoptysis.	Father emphysematous, died, æt. 70, of cerebral softening. Mother died, æt. 38, was said to be ataxic, like her daughter.	*The patient*, a female, died of phthisis. Table II., 22. No note whether she had brothers or sisters.
5. Carré, as quoted by Brousse:— *Grandmother* (maternal?) *was ataxic.*	*Mother ataxic.* *Mother's brothers and sisters,* 8 *in number, all ataxic.*	There were 12 children, of whom 5 were not affected, 7 *were ataxic*, viz. the patient (female). Table II., 21. 4 brothers, 2 sisters. *One cousin* (*male*) *was also ataxic.*

[1] We omit from these tables the case by Kahler and Pick, which is chiefly of anatomical interest; those by Seeligmüller, which Friedreich did not admit to be cases of hereditary ataxy; those by Kellogg and by Schmid, the original account of which we have not been able to obtain; and one or two cases quoted by Hammond.

TABLE I.—To show the Genealogical Relations of most of the Published Cases (*continued*).

Rutimeyer.

6. Blattner Family.—Four families, in each of which there were cases of the disease, were descended in the male line from a common great-great-great-grandfather (who was nicknamed the "Stumbler") as follows;—


Blattner (A), nicknamed "Stulpi," presumably ataxic?
(B)
(b)
(c)
(d)
(C)
(C1)
(D)
(D1)
(E) Blattner m. Blattner, had 10 children, viz. 7 healthy, 3 diseased.
(E1) Blattner m. Kyburg, had 7 children, viz. 6 healthy, 1 diseased.
(E2) Blattner m. Wehrli, had 10 children, viz. 7 healthy, 3 diseased.
(e) Blattner m. Basler, had 7 children, viz. 6 healthy, 1 diseased.

See Table II., 10-17. Of the 8 ataxics, 6 were males, 2 females.
In the case of the Blattner-Basler branch there were nervous complaints (chorea, poliomyelitis) on the mother's side.

Quincke and Rutimeyer.

7. Kern Family:—

Father's family and father quite healthy. Mother's father intemperate, and during last part of his life mentally affected. — Mother suffered from "general nervousness." — There were 10 children, of whom 7 were healthy, 3 *were diseased*, 1 male, 2 females. Table II., 18-20.

Carpenter.

8. Father healthy. Father's brother died of general paralysis. Father's cousin peculiar, hysterical? Mother (and her family) healthy. — There were 8 children, of whom 5 were healthy, 1 healthy at the time of Dr. Carpenter's observations, 2 *were diseased*, females. Table II., 23, 24.

	Gowers.			
9.	SHAW FAMILY:— Father's mother was paralysed in her tongue.	Father died of Bright's disease. Father's brother ditto. Another brother died insane. Mother died late in life of abdominal tumour; in early life had two attacks of chorea.	There were 9 children, of whom 1 died, æt. 10 years, 3 (males) were healthy, 5 *were diseased*, viz. 4 males and 1 female. Table II., 29–34.	
	Dreschfeld.			
10.	Father, æt. 72, living and healthy. Mother died, æt. 54, of bronchitis.	There were 15 children, of whom 10 were not affected (3, however, died quite young). Two of these 10 were married, and had healthy children. 5 *were diseased*, 3 males and 2 females (viz. Table II., Cases 25–27, and two who had other patients died previously).		
	Hammond.			
11.	No disease of the kind known in previous generations.	*Two brothers were diseased.*		Table II., 35, 36.
12.	No neurosis known in the family.	Ditto.	ditto.	Table II., 37, 38.
13.	Ditto. ditto.	Ditto.	ditto.	Table II., 39, 40.
	Wickham Legg and D'Arcy Power.			
14.	In grandparents and other ancestry there was no nervous disease known.	Father healthy, and has 3 brothers living, healthy, 1 brother dead of small-pox. 2 sisters living, healthy, 1 sister dead of phthisis. Mother healthy, and all her brothers and sisters healthy.	There were 10 children, of whom 7 were healthy, 3 *diseased*, viz. the patient, a male, the eldest. Table II., 34. One brother (dead) and one sister, who, according to the patient, suffered like himself.	

TABLE II.—Abstract o

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition; and γ, electro-irritability of muscles. δ, Contractures; ε, tremors, η, choreic movements.
1	Andreas Lotsch (Friedreich, Case 1).	18	Difficulty in walking.	19 years. [watched for 2 years, when death from typhoid.]	Cannot stand or sit upright. Head oscillates. Cannot lift cup to mouth, or take hold of an object easily.	Can move lower limbs, but with effort; adductors of thighs paralysed. Muscular power of upper limbs normal. Muscles act to electricity.
2	Charlotte Lotsch (Friedreich, Case 2).	18	Weakness, first of left then of right leg.	13 years, 31 years [and for 2 years till death from nephritis].	Cannot stand without support. Cannot pick up things, nor thread a needle. Later, oscillations of the head.	Movements of lower limbs performed with effort; finally paralysis, with contracture of some muscles, atrophy of others. Upper limbs, no paralysis.
3	Justinia Süss (Friedreich, Case 3).	16	Weakness and wandering pains, in legs.	15 years.	Gait ataxic: needs support, even for standing. Fine movements of hands difficult. Slight oscillations of head and neck.	Simple movements performed with effort, but electro-contractility of muscles normal. Had had involuntary flexions of lower limbs, with cramp.
4	Salome Süss (Friedreich, Case 4).	14	Curvature of spine, weakness and pain in legs 2 to 3 years later.	14 or 12 years.	Cannot stand or sit upright. Cannot button clothes nor thread needle.	Simple movements performed feebly and with effort. Electro-contractility of muscles normal.
5	Lisette Süss (Friedreich, Case 5).	15	Weakness of legs, then of arms. "Gnawing" pain in bones.	17 years.	Bedridden for years. Movements of hands ataxic. Head and trunk oscillate.	Simple movements require much effort.
6	Friedrich Süss (Friedreich, Case 6).	15	Weakness of left leg and arm, then of right.	9 years. 12 years. 25 years.	Gait that of a drunken man. Totters as he stands. Worse in dark or with eyes shut. Fine movements of hands difficult. Oscillations of head late in the disease.	Muscular power and electro-contractility normal.

INDIVIDUAL CASES.

Sensory system. Sensibility to touch, pain, and temperature; muscular sense; electro-muscular sensibility; shooting-pains, girdle pains, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
Normal, except for a diminution of electro-sensibility in the muscles. No pains.		Normal.	Speech stammering and difficult to understand. Fine tremor of tongue.	Normal.		Normal.
Normal. Cramps and pains in lower limbs, towards the later period of the disease.	Tendon-reactions absent. Skin-reflexes present.	Kypho-scoliosis becoming worse.	Speech thick and stammering, finally unintelligible.	Nystagmus late in the disease.	Polyuria, salivation, &c. Some bladder difficulty (later).	Occasional vertigo.
Tactile sensibility diminished in lower limbs and abdomen. Electro-muscular sensibility diminished. *Occasional pains*, sometimes wandering, sometimes shooting, from earliest period.		Kypho-scoliosis said to have appeared early.	Speech stammering and often unintelligible.	Nystagmus.		
Electro-muscular sensibility diminished. Occasional shooting *pains* from early period, and cramps.		Kypho-scoliosis.	Speech heavy and stammering; intelligible.	Slight nystagmus.	Palpitation (systolic murmur). Cough and dyspnœa.	Frontal headache.
Pains in legs frequent: gnawing pains an early symptom; later "neuralgic" pains; finally, painful cramps in various parts.		Kypho-scoliosis.	Speech affected.	Normal.	Palpitation and dyspnœa.	Had had hysterical attacks. Recently vertigo.
Normal in every way.			Speech heavy and stammering, finally unintelligible.	Slight nystagmus late in the disease.	Loss of sexual power.	Vertigo.

TABLE II.—ABSTRACT OF

No. in present Table.	——	Age when first attacked.	Symptoms when first attacked.	Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition; and γ, electro-irritability of muscles. δ, Contractures; ε, tremors, η, choreic movements.
7	Louise Schulz (Case 7, Friedreich).	13	Stumbling gait, clumsiness of hands (headache 2 years before).	5 years. 9 years. [Watched for 3 years each time.]	Well-marked ataxia of movement, both in upper and lower limbs; not increased by closure of eyes. (Later, inability to stand or sit without irregular movements of head and trunk.)	No loss of power. Muscles natural. Later, slight twitching of finger, hand, &c.
8	Katharina Schulz (Case 8, Friedreich).	13	Helplessness of legs, then of arms (headache previously).	3 years. [Watched for 2 years more.]	Similar to last case (but symptoms rather less marked, and not progressing while under observation).	Normal.
9	Marie Schulz (Case 9, Friedreich).	13	Upper and lower limbs affected at nearly the same time.	1 year. 4 years.	As in the two last cases. (Later, staggered during standing.)	Normal.
10	Rudolf Blattner (Case 1, Rutimeyer).	7	Uncertainty in walking and standing (especially in the dark).	10 years.	Walks with feet apart, staggering. Sways as he stands. Worse with eyes shut. Difficulty in buttoning clothes, &c. Slight oscillation of head and trunk as he sits.	No paralysis. Muscles on ulnar side of right forearm slightly wasted. Some irregular movements when he holds out arms. Slight flexion of some of the fingers, and dorsal flexion of great toes.
11	Gottlieb Blattner (Case 2, Rutimeyer).	7	Weakness and uncertainty in walking.	27 years.	Ataxic stage had passed into that of paralysis when patient came under observation.	Lower limbs almost paralysed. Muscles atrophied. Upper limbs paretic atrophied. Contracture of some fingers, flexion of knees, double equino-varus, dorsal flexion of great toes.
12	Marie Blattner (Case 3, Rutimeyer).	6	As in the two last cases.	23 years.	Unable to walk. Writing, buttoning clothes, &c., very awkwardly performed.	Lower limbs paretic, not wasted. Contractures of fingers and hands, slight equino-varus, dorsal flexion of toes.
13	Rudolf Blattner, β. (Case 4, Rutimeyer).	7	Fatigue and uncertainty in walking, especially in the dark.	8 years.	Walks with feet apart, staggering. Swaying of trunk as he stands. Worse with eyes shut. Complicated movements of hands awkwardly performed.	Nutrition normal, except that left side of face is less developed than right. Slight dorsal flexion of great toe.

INDIVIDUAL CASES—*continued.*

Sensory system. Sensibility to touch, pain, and temperature; muscular sense; electro-muscular sensibility; shooting-pains, girdle pains, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
Quite normal. [Later, slight diminution of sensibility to touch, and to electricity in the soles?]	Tendon reactions absent. Skin-reflexes normal.	Normal.	Words uttered slowly and with abrupt pauses. Movements of tongue and palate normal.	Normal.	Intercurrent albuminuria (? nephritis).	Headache (migraine?) preceded onset of disease.
Normal.			Speech affected.	Normal.		Do.
Normal.	Tendon-reactions absent. Skin-reflexes normal.		Speech affected soon after limbs.	Nystagmus when last observed.	Normal.	Normal.
Slight diminution of tactile sense in lower limbs (?), otherwise normal.	Tendon-reactions absent. Skin-reflexes normal.	Slight scoliosis.	Speech drawling, nasal, with frequent change of pitch. Slight tremor of tongue.	Nystagmus developed when under observation.	Slight systolic apex murmur. Subject to hiccough.	Normal.
Tactile sense diminished in both upper and lower limbs. Conduction of pain delayed.	Tendon-reactions absent. Skin-reflexes normal.	Marked kyphosis and scoliosis.	Speech slow, stammering, almost unintelligible.	Nystagmus. Deafness.	Frequent sweatings and salivations. Hiccough on drinking.	Normal?
Tactile sense diminished in upper and still more in lower limbs.	Tendon-reactions absent. Skin-reflexes normal.	Marked kyphosis and scoliosis.	Speech drawling, but intelligible.	Nystagmus. Sight and hearing said to have been affected.	Cyanosis and œdema of legs and feet.	Normal.
Normal. (Particularly sensitive to electric current.)	Tendon-reactions absent. Skin-reflexes normal.	Slight kyphosis and scoliosis.	Speech drawling, somewhat nasal.	Nystagmus developed while under observation.		

TABLE II.—ABSTRACT OF INDIVI

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition; and γ, electro-irritability of muscles. δ, Contractures; ε, tremors, η, choreic movements.	Se Sens pain, muscu muse sha pa
14	Karl Blattner (Case 5, Rutimeyer).		Never learnt to walk straight. Gait more uncertain last few years.	Boy (age not stated).	Walks in zig-zags. Difficulty in complicated movements. "Writes over the lines." Gait worse with eyes shut. Jerking movements of head.	Twitchings of fingers and of isolated muscles when he holds out arms.	Norm sen in t
15	Maria Magdalene Blattner (Case 6, Rutimeyer).	4 or 5	Arms and legs simultaneously affected. Speech not till 8 or 10 years later.	15 years.	Can only walk with support; throws legs about. Complicated movements of hands and arms uncertainly performed. Symptoms not worse when eyes are shut.	Paresis of lower limbs. Dorsal flexion of great toes.	Tacti ish cas tio
16	Jacob Blattner (Case 7, Rutimeyer).		As in previous case. Disease followed chorea.	Not seen by author.		Paralysed like Gottlieb. No. 11.	
17	Fritz Blattner (Case 8, Rutimeyer).	4	Uncertainty in walking.	Not seen by author.			
18	Heinrich Kern (Case 9, Rutimeyer and Quincke.)	6 or 7	Ditto.	6 years and 13 years.	Waddling gait. Head and trunk sway as he stands. Worse with eyes shut. Ataxia of arms less than of legs, not worse with eyes shut.	At time of second observation, almost complete paralysis of lower limbs. Wasting of muscles, esp. below knees. Contractures at knees; double equino-varus: dorsal flexion of great toes.	Tact ed lo sli pe al in
19	Hedwig Kern (Case 10, Rutimeyer and Quincke.)	7 or 8	Ditto.	8 or 9 years.	Jactitation of legs in walking. Needs support. Head and trunk sway as she stands. Not worse with eyes shut. Movements of arms very uncertain.	No paralysis. Slight wavering of arms when held out. Dorsal flexion of great toes (during last year).	Tact di lo
20	Bertha Kern (Case 11, Rutimeyer).	7	Ditto.	6 years.	Much as in last case, though rather less marked.	No paralysis. Slight dorsal flexion of great toes.	Tact di be lo

INDIVIDUAL CASES—*continued.*

Sensory system. Sensibility to touch, pain, and temperature; muscular sense; electro-muscular sensibility; shooting-pains, girdle pains, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
Normal. (Tactile sense diminished in thighs?)	Tendon-reactions absent. Skin-reflexes lively.	Slight kyphosis and scoliosis.	Speech slow, drawling, and nasal.	Nystagmus.		
Tactile sense diminished in legs. Occasional formication.	Tendon-reactions absent. Skin-reflexes normal.	Slight kyphosis.	Speech drawling, clumsy. Fibrillary tremor of tongue.	Nystagmus.	Cyanosis and œdema of feet.	
			Affected.			Chorea at first. Fits, in one of which he died, æt. 18.
			Not affected (?) but died at 9 of measles.			
Tactile sense markedly diminished in lower limbs, slightly so in upper. (At first only a little formication in feet.)	Tendon-reactions absent. Skin-reflexes normal.	At first normal; later marked kyphosis and scoliosis.	Speech indistinct, tongue cannot be held still. Afterwards speech worse, only slight tremor of tongue.	Nystagmus (at second observation).	Cyanosis of feet, slight œdema.	Vertigo among the early symptoms.
Tactile sense slightly diminished (?) in lower limbs.	Tendon-reactions absent. Skin-reflexes normal.	Scoliosis (during last year).	Somewhat drawling. No tremor, &c., of tongue.	Slight nystagmus.		
Tactile sense slightly diminished (?) in both upper and lower limbs.	Tendon-reactions absent. Skin-reflexes normal.		Speech not affected.	Slight nystagmus.	Some coldness and cyanosis of legs and feet. Obscure affection of knee at age of 5.	

TABLE II.—Abstract of

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition; and γ, electro-irritability of muscles. δ, Contractures; ε, tremors; η, choreic movements.
21	Carré's case, female, as quoted by Brousse.	22	Pains in feet and legs; then numbness and difficulty in walking.	6 years.	Peculiar gait; short, jerky steps. Disorderly movements of head and eyes as she walks. Not worse with eyes shut. Fine movements of hands disorderly.	No paralysis.
22	Brousse's case, female.	24	Weakness and incoordination of legs in walking.	8 years.	Can walk only with support: throws feet forward and outward. Difficulty in picking up pins, &c. Worse with eyes shut; cannot stand. Occasional tremor of head.	No paralysis. Swaying movements of arms when stretched out.
23	Female (Case 1, Carpenter).	12	Unsteady gait. Spinal curvature.	17 years (at time of report).	Gait uncertain, as if intoxicated: foot brought down forcibly, heel first. Some clumsiness of hands. Worse with eyes shut.	No paralysis. Electro-contractility diminished below knees. Tendency to talipes equinus.
24	Female (Case 2, Carpenter).	12	Spinal curvature. Irregular gait 2 years later.	15 years (at time of report).	Similar symptoms (but less marked) to those in last case. Sight less necessary to her. Head and shoulders shaky.	Electro-contractility (in legs) below normal.
25	Male (Case 1, Dreschfeld).	20	Weakness in walking; esp. in dark. Shooting-pains.	22 years.	Fine movements of hands difficult. Ataxy of legs merging into paralysis.	Simple movements of legs (when recumbent) slow and jerky. Paresis of legs. Grasp weak. Muscles flabby.
26	Male (Case 2, Dreschfeld).	23	Weakness of knees. Pain in lower limbs.	9 years.	Jactitation of feet as he walks. No ataxia of upper limbs. (At first, difficulty of walking in dark.)	Atrophy of muscles of hands, forearms and legs. Grasp weak. Diminution of electro-contractility. (Muscles of face slightly atrophied.)
27	Female[1] (Case 3, Dreschfeld).	20	Sharp pains. Heaviness in walking.	14 years.	Ataxic gait: cannot walk with eyes shut. No ataxia of arms.	Muscles of thighs somewhat somewhat flabby, but no paresis.

[1] Two other members of the same family appear

INDIVIDUAL CASES—*continued.*

Sensory system. Sensibility to touch, pain, and temperature; muscular sense; electro-muscular sensibility; shooting-pains, girdle pains, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses.)	Visceral vaso-motor secretory functions.	Cerebral functions.
Pains in early part of disease, but sensory functions proved quite normal.			Speech stammering, as if tongue were fixed for a moment. Tremor of tongue when put out.	Diplopia and supposed imperfection of sight.		
Normal.	Tendon-reactions absent.		Speech hesitating and sputtering. Movements of tongue normal.			Convulsions in infancy. Intelligence low; irritable, &c. Died in fit (subarachnoid hæmorrhage).
Slight and occasional numbness in legs; tactile sensibility slightly lowered? Electro-muscular sensibility normal.	No plantar reflex.	Lateral curvature.	"Precision in speech," as if effort to co-ordinate.	Normal.	Skin of legs easily frozen by ether-spray.	Normal.
Normal.		Lateral curvature.	Speech hesitating as if there was something under tongue.	Normal.	Ditto.	Normal.
Tactile sensibility much diminished in lower limbs. Pains at onset of disease.			Normal.	Normal.		
Electro-muscular sensibility diminished in legs. Occasional numbness, but no loss of tactile sense. Pains at onset.			No abnormality mentioned.	Vision impaired at first (since normal).	Loss of sexual power.	
Tactile sensibility somewhat diminished in legs (pains at onset).			Normal.			

to have been affected in a similar fashion.

TABLE II.—ABSTRACT OF

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition; and γ, electro-irritability of muscles. δ, Contractures; ε, tremors; η, choreic movements.
29	Male (Case 1, Gowers).	21	Unsteady walk.	18 years.	Gait not remarkably ataxic; legs lifted too high if he takes long steps.	Some weakness of legs.
30	Female (Case 2, Gowers).	18	Difficulty in walking.	11 years.	Difficulty in standing out of proportion to muscular weakness. Ataxy of arms. Head steady.	Ditto.
31	Male (Case 3, Gowers).	19	Ditto (?).	4 years.	Takes short steps and sways about. Head unsteady. Can only stand with feet apart. Arms ataxic: can barely write.	No paresis.
32	Male (Case 4, Gowers).	20 (?)	Slight unsteadiness.	2 or 3 years (?).	Difficulty in standing with eyes shut. Arms natural.	
33	Male (Case 5, Gowers).	?		?	Unsteadiness in turning. Cannot stand with eyes shut. Handwriting unsteady.	No paresis.
34	Power's case. Male.	15	Loss of power in legs. Vomitings.	7 years.	Cannot even sit upright. Head nods and falls forward. Buttons clothes with difficulty; cannot do it with eyes shut.	Wasting of legs (and thighs?). farado-contractility normal. Extension of legs performed with efforts. Legs jump at night. (Wasting of each 5th metacarpo-phalangeal joint.)
35	Male (Case 1, Hammond).	4	Fell when walking.	5 years. 15 years.	At first observation could not stand; at second, could not sit up.	All four limbs said to be "paralysed," but legs can be moved as he lies down.
36	Male (Case 2, Hammond). Brother of last.	10½	Weakness of legs.	6 months. 10½ years.	"Gait staggering, but not like locomotor ataxy." Not worse with eyes shut. At date of second observation could not walk without help. Arms affected.	

INDIVIDUAL CASES—*continued.*

Sensory system. Sensibility to touch, pain, and temperature; muscular sense; electro-muscular sensibility; shooting-pains, girdle pains, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
Sensibility to pain increased; otherwise no abnormality.	Tendon-reactions absent. Skin-reflexes mostly active.		Syllables run together, sometimes unduly separated.			
Normal.	Tendon-reactions absent.		Speech markedly syllabic.			
Normal.	Tendon-reactions absent. Skin-reflexes mostly active.		Articulation confluent.			
Tactile sensibility impaired in legs. Otherwise no abnormality.	Tendon-reactions absent. Skin-reflexes lessened.		Tendency to confluent articulation.			
Ditto.	Tendon-reactions absent. Skin-reflexes mostly present.		Articulation sometimes syllabic, sometimes confluent.			
Bouts of shooting-pain in back. Slight impairment of tactile sensibility in legs. Numb feeling in arms and legs.	Tendon-reactions absent. Skin-reflexes normal.	Lateral curvature (lumbar).	Speech jerky, sometimes slow, sometimes rapid, slurring. Difficulty in mastication, and swallowing.	Optic discs rather white, otherwise normal.	Vomitings (at first). Some incontinence of urine.	Weak intellect. Had attacks of giddiness with loss of consciousness and falling.
Normal.	Skin-reflexes normal.		Speech barely intelligible.	Normal.		Normal.
			Speech indistinct at date of second observation.			

TABLE II.—ABSTRACT

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition; and γ, electro-irritability of muscles. δ, Contractures; ε, tremors; η, choreic movements.
37	Male (Case 3. Hammond).	6	Stumbling. Fatigue in walking.	4 years.	Cannot walk without help. Gait said to be that of paralysis, not ataxy. Not worse with eyes shut. Head falls forward as he sits.	"Upper limbs weak, so that he cannot feed himself." Nutrition normal. (Muscular spasms before death.)
38	Male (Case 4, Hammond).	8	Weakness in legs.	Course of disease like his brother's (last case).		
39	Male (Case 5, Hammond).	3	Weakness first of right then of left leg.	13 years. 27 years.	Gait staggering: sways a little with eyes shut. Later cannot stand with eyes shut.	Movements of legs made with effort. Hands weak. Electro-contractility normal. Later, involuntary twitchings of hands.
40	Male (Case 6, Hammond).	7	Weakness of legs.	19 years.	Symptoms as in last case (his brother), only less pronounced.	

INDIVIDUAL CASES—*continued.*

Sensory system. Sensibility to touch, pain, and temperature; muscular sense; electro-muscular sensibility; shooting pains, girdle pains, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
Normal. (Severe pains before death.)			Speech barely intelligible. Difficulty in swallowing.			Frequent vertigo. Mind ill-developed.
No pains till just before death.						
Pains back of head and neck (on second observation.	Tendon-reactions absent.		Difficulty in pronouncing words: often forgets them.	Nystagmus (on second observation). Sight bad at night.		Vertigo.
	Tendon-reactions absent.			Deaf left ear, from measles.		

Reviews and Notices of Books.

Recherches sur le Système Nerveux des Poissons.—Par EMILE BAUDELOT, professeur à la Faculté des Sciences de Nancy. Ouvrage précédé d'un avertissement par M. EMILE BLANCHARD, et accompagné de 10 planches gravées. Paris; in folio; 1883. G. Masson.

THIS very remarkable and important work is a posthumous one, and has been published by the care of Madame E. Baudelot, the widow of the distinguished scientist, and M. E. Blanchard, the well-known naturalist, and president of the Academy of Sciences in Paris. Although the author died before having investigated the subject as thoroughly as he intended to do, this book may be considered as presenting a fair sketch of what it would have become if the author had lived. Baudelot had written a good deal on fishes generally, and had studied their nervous system very accurately. The subject had been rather overlooked: little was known concerning that question. In the present review we must leave the greater part of the book untouched, and confine ourselves to the chapter dealing with physiological questions. Concerning the functions of the central nervous system, M. Baudelot has made some interesting experiments. As Desmoulins and Magendie have already shown, the loss of the cerebral hemispheres does not interfere with the power of motion, nor with any special function. Fishes move about as easily and as unconcernedly as if nothing had been done to them.

The loss of the superior part of the optic lobes does not affect fishes any more than that of the cerebral hemispheres in regard to movement, but the eyesight seems impaired, the fish remains quiet, as if it were asleep. If the inferior part of the optic lobes is destroyed or injured in some manner, a great change in the movements is noticed. The fish turns round and round on the long axis of its body; it falls on the side opposite to the injured one, and goes on spinning in the same manner during many days, sometimes a fortnight. This rolling motion may be executed ten, twenty, even a hundred or more times per minute. When it stops, the fish lies on the side opposite to the injury; the body is also more or less bent up on the same side (*mouvement de manège*). Baudelot believes that the rolling motion (*mouvement de rotation autour de l'axe*) may be ascribed to a painful feeling of contracture.

If the medulla oblongata is injured, there is also a rolling motion, but quite a different one: the motion is contrary to that

of the preceding experiment: the fish lies on the injured side. The injuries of the cerebellum do not seem to affect motion nor sensation, unless the medulla oblongata is also injured.

Generally speaking, injuries of the cerebral hemispheres do not affect motion, nor sensation, nor intellect in fishes, while they do affect voluntary motion and intellect in mammalia; the injuries of the cerebellum impair motor co-ordination in mammalia, but do no harm to fishes. Injuries of the superior part of the optic lobes affect fishes, as those of the corpora quadrigemina affect mammalia; injuries of the inferior part of the same lobes affect fishes, as those of the optic thalami and cerebral and cerebellar peduncles affect mammalia.

Although the physiological part of Baudelot's work is not a very long one, it is of much importance in the study of cerebral physiology generally.

Of the remainder of the book we shall only say that it is full of interesting information: it was not to be expected that there could be in the cerebral form of the different sorts of fishes the variety that M. Baudelot shows to exist.

The engravings are excellent: they add a great deal to the value of this book, and help us greatly to understand the general morphology of the brain, as explained by the much regretted and distinguished zoologist. Mme. Baudelot deserves much praise for not having allowed her husband's valuable researches to be lost; M. E. Blanchard and G. Masson are also to be complimented for their generous attachment to science which induces them, especially Mons. G. Masson, to spend much time and assume much trouble in pursuits that are generally known to be more costly than profitable.

HENRY DE VARIGNY.

Clinical Lectures on Mental Diseases. By T. S. CLOUSTON, M.D. &c. London: J. and A Churchill; 1883. Crown 8vo. pp. 631. Illustrated.

MR. G. T. ROMANES has lately published two books. In one of these he narrates a vast number of instances of intelligent conduct on the part of animals; in the other he investigates deeply the constitution of mind and the workings of the nervous system, and deduces certain broad general principles which he elucidates and illustrates by means of the facts contained in his first volume. The first book is a storehouse of data, a granary of facts, a museum of instances, a treasury of records, a warehouse of raw material: the second is the manufactured article, it is the history which the records disjointedly narrate, it gives the laws to which the instances apply, it draws the conclusions from the data, it displays the utilisation of the facts. The first book is purely

narrative, the second mainly argumentative. The danger in the second book is of fallacy; the danger in the first is of "anecdotage."

Dr. Clouston's book fulfils, in a different province, nearly the same function as Mr. Romanes' first work on Animal Intelligence. It is one long record of accurate and laborious observations. From cover to cover the book is positively bursting with facts. It presents to the student of alienism an almost embarrassing profusion of data. The number of cases which are recorded in full is considerable; and the skill with which the salient points of each case have been chosen for presentation, and the graphic terseness with which they are described, will be fully appreciated by those only who have attempted similar tasks with less success. The records of cases are far from being the only materials placed at our disposal in this interesting volume. Repeatedly at frequent intervals the results of Dr. Clouston's ripe experience are put before us in the form of empirical generalisations, which, representing multitudinous observations, are equivalent to large groups of facts.

Nor is this all, for in addition to the excellent therapeutics with which the book is saturated, we meet with many shrewd remarks, and occasionally with a flash of true philosophic insight. A few samples will whet the reader's appetite for more. "There is no use," says Dr. Clouston, "in dunning a patient to 'rouse yourself,' to 'throw off your dulness,' to 'drop those fancies,' for in many cases it would be just as wise to tell a hemiplegic to 'move that leg.'" "We think of melancholia chiefly from the patient's subjective point of view, taking his affective change and his conscious mental pain chiefly into consideration; while we think of mania more from our own objective point of view, and picture the patient's talkativeness, his restlessness, and his manifest changes of personality and habits." "The ravings of a maniacal patient are often well worthy of study. . . . There is no such thing as real incoherence. The words and ideas always cohere by some bond or other." The homicidal feeling "is undoubtedly the human instinct of slaughter and destruction." It "is analogous to the physiological instinct of breaking things in children." It is no doubt a case of "throwing back," of atavism. It is a reappearance in excess of the pugnacious instincts of our savage ancestors. "Why," asks Dr. Clouston, speaking of the ill-effects of high-pressure education upon growing girls, "why should we spoil a good mother by making an ordinary grammarian?"

Like every other writer upon insanity, Dr. Clouston proposes his own classification; of which it may be said that nothing would be more easy than to pull it to pieces and display its inconsistency, nothing more difficult than to construct a better. In the chapter on Epilepsy, Dr. Clouston exhibits a curious but by no means unique misapprehension of Dr. Hughlings-Jackson's views on this subject, the doctrines attributed to Dr. Jackson being such as he would shudder to hear of.

While acknowledging the admirable manner in which Dr.

Clouston has accomplished the task to which he committed himself when he undertook to write a book of Clinical Lectures on Insanity, it is right to point out the limitation of that task. Of the science of Insanity, as of every other science, there are two aspects; and of these Dr. Clouston limits himself to one. Nothing could be clearer than the way in which he presents that aspect of the subject with which alone he undertakes to deal; but it is necessary to remember that this is only one of two aspects, and the view that he gives us is unavoidably imperfect. He has given us knowledge; but knowledge is not science until it is organised; and the organisation is wanting. To render the work complete we want a book that shall treat the subject from the opposite point of view, and be to Dr. Clouston's present work what Mr. Romanes' second book is to his first. We have the facts; we now want the principles. We have the data; we now want the conclusions. We have the instances; we now want an enunciation of the laws to which they conform. We want an account of the workings of the normal mind and of the normal nervous system; an explanation of the ways in which it is possible for them to become disordered; a description of the results of such disorder, illustrated by the instances which Dr. Clouston's book affords in such abundance. Facts accumulate upon us, until we stagger under the burden of them, and are fain to tumble them down here and there in disorderly heaps. Our most urgent need is of an architect who will provide some plan upon which we can build them up into a regular and useful structure.

CHARLES MERCIER, M.B.

A Practical Introduction to Medical Electricity. By A. DE WATTEVILLE, M.A., M.D., B.Sc., &c. Physician in charge of the Electrical Department at St. Mary's Hospital, London, &c. Second Edition. London: H. K. Lewis, 1884. 8vo. pp. 220; with 89 woodcuts, and five plates of the motor points.

THE employment of electricity in practical medicine has encountered vicissitudes so great and of such variety, as make it a matter of some wonder that anything satisfactory survives the trying ordeal to which it has been exposed. Beyond the well-meaning, but self-deceived enthusiasts who have published exaggerated accounts of its utility, and the impudent quacks who have traded on the mystery of its force, there have, however, happily been cautious and persevering observers whose communications have been always tending to bring order out of chaos; and we may hope that a time is within sight when the place of the electric current in the practice of our profession will be more or less definitely fixed.

In his earlier edition (which was noticed three years ago in these pages) the author rendered excellent service to the cause of progress by giving us, what had previously to a great extent been wanting in English works upon electro-therapeutics, solid and

accurate information upon the theory of electricity, so far as concerned us as physicians. The present condition of his work is so enlarged, and for the most part re-written, as to constitute a new book. In it the author has still further endeavoured to popularise such elementary facts in electrophysics as will go far, if mastered by the reader, to preserve him from misconception, and keep him upon the right track for a really scientific observation of such phenomena as present themselves to him when using electrical currents in the practice of medicine. Dr. de Watteville still insists upon measurements of current strength as the essential condition of a rational application of electricity to medicine, and advocates the constant employment of the galvanometer. We would direct especial attention to the part of his book in which he deals with this question. It contains much valuable information and judicious advice. So also, as regards the question of electrical distribution or diffusion, we think he makes a decided point in suggesting the adoption of Professor Erb's set of standard electrodes, so as to fix, once for all, the meaning of the terms "small," "large," "moderate sized," so frequently used. The relative position of the electrodes upon the body in various applications of currents is considered very fully, and the diagrams borrowed from Erb, which are introduced to illustrate the subject, are clear and eminently descriptive. Nearly 100 pages are devoted to these and other questions in electrophysics, together with descriptions of the apparatus most serviceable in medical practice.

Chapter II. deals with electro-physiology. A novel feature is here introduced. The instruction is given in the form of experiments which the reader may perform upon himself. These are numbered, and arranged for reference, one to another. This is an excellent method, by which a thoroughly practical idea of the subject may be obtained, and a variety of erroneous notions dispelled.

We have been disappointed that Dr. de Watteville has not preserved the German notation for marking qualitative changes in the excitability of nerve and muscle. As we have before suggested, if the word "shut," be used instead of "close," as regards the circuit, and the letter Z (Zuckung = con'raction) be accepted as an arbitrary sign implying contraction (and there is something in the form of the letter suggestive of it), records made by English and most foreign observers will be mutually intelligible. Faraday introduced the terms, "anode" and "kathode," which have been adopted by the Germans. In this country instead of the letters K S Z for "kathodic shutting contraction," the letters C C C, "cathode closure contraction," have just got to be pretty generally used, when Dr. de Watteville again disturbs the harmony by employing K instead of C, so that now we have K C C. Surely amongst all these diversities we are in danger of losing sight of the Q. E. D.! The adoption of the German notation would have saved all this inconvenience, and it is, we think, to be desired that English-speaking observers may even yet be led to see the advantage of it.

The subject of electro-diagnosis is considered in Chapter III., and the numerous difficulties and sources of fallacy in investigations are pointed out in a very graphic manner. Chapter IV. is devoted to electro-therapeutics. In it the author shows that he is anxious that the reader should clearly understand the rationale of the methods of applying the current to the human body.

The work is well illustrated by numerous engravings, amongst which the plates showing the motor points strike us as exceptionally clear. There is no doubt that this book, based as it is upon all the recent advances in electrical science, must take a most important place amongst works in English upon the subject of electricity as applied to medicine, and we congratulate the author on his achievement of a difficult task.

T. Buzzard, M.D.

Clinical Lectures on Giddiness. By Thomas Grainger Stewart, M.D., Professor of the Practice of Physic in the University of Edinburgh, &c. 8vo. pp. 43. Edinburgh, 1884.

This pamphlet consists of three clinical lectures devoted to the discussion of the symptom giddiness. The subject is treated in a comprehensive manner, and at the same time in a concise and intelligible form. The author, after a short description of what is understood by the term, proceeds to investigate its relation to disorders of the sensory and motor functions. It is explained that equilibrium is not a condition born with the individual, but is acquired as the result of education, and is thereby subservient to the tactile, ocular, muscular, articular, visceral, and labyrinthine impressions. It is pointed out that the semi-circular canals are the chief sensory organs presiding over this function, and for its proper performance there must be integrity of these as well as of the nervous centres, and the conducting nerve fibres, change in any of which may lead to abnormal modifications, which among other symptoms are manifested by vertigo. In health all the peripheral and central conditions harmonise to preserve normal equilibrium; where there is any break in this arrangement, or where there is "a contradictoriness of sensory impressions," giddiness is the result. This may be due to peculiarities in external conditions, and more especially through the medium of the sight, as evidenced by the peculiar sensations experienced when we look from a height or at a distorted object, such as the Tower of Pisa, or in waltzing, swinging, or being on board ship. Again, giddiness may be due to abnormalities of the sensory structures themselves, as seen in strabismus, diplopia, nystagmus, &c. The most important form of vertigo is that resulting from causes situated in the semi-circular canals, and which is known as Menière's disease. Various varieties of this have been described, as the external, middle, or internal ear

is affected. It is pointed out that giddiness may arise from diseases of the spinal cord as in locomotor ataxy, multiple sclerosis, &c., and may be explained partly by the defective action of the muscles of the eye, and partly from changes in other sensory impressions. Vertigo may arise from a variety of intra-cranial abnormalities, and amongst those especially referred to are those localised in the cerebellum, crura cerebelli, pons, cerebral peduncles, and corpora quadrigemina. Besides these the symptom may be the result of various diseases of the brain, either organic or functional, such as tumours, congestion, anæmia, injuries, epilepsy, megrim, disease of the arteries, effects of galvanism, &c. A variety of morbid conditions of the general system may be followed by the same derangement as in nervous debility, dyspepsia, toxic infection, fevers, constitutional cachexias, and so on. The explanation of the occurrence of giddiness as a result of these is somewhat doubtful, but is probably to be explained by their effects on the blood, and its abnormal influence on the nerve centres.

Contribution à l'étude et au diagnostic des formes frustes de la Maladie de Basedow. Par M. le Dr. PIERRE MARIE. Paris, 1884.

THE author of this little book does not claim that it is a complete monograph on the subject of Basedow's disease. It professes only to discuss a certain number of those marked symptoms which are not usually described in association with the malady, or which have been considered as purely accessory. These, on the contrary, M. Marie believes to be intimately connected with the affection, constituting peculiar and characteristic features, sufficient to enable us to detect the disorder, even when the special symptoms of goitre and exophthalmus are wanting. The general conclusions arrived at are—

1. Tremor is a frequent symptom of Basedow's disease, and is probably almost always present.

2. This tremor has special characters which permit it to be distinguished from others.

3. The presence of goitre or exophthalmus, either together or singly, are not essential elements of the disease.

4. When this tremor accompanies palpitation of the heart, without notable augmentation of the temperature, Basedow's disease may be suspected. The diagnosis will be confirmed by the presence of one or more of the following symptoms:—Local or general sweating, boulimia, insomnia, cough without expectoration, or abnormal physical signs in the chest.

A. HUGHES BENNETT, M.D.

Abstracts of British and Foreign Journals.

Ferrier and Yeo on the Effects of Lesions of the Cerebral Hemispheres. (*Proceedings Royal Society*, xxxvi. 229.)—The following is an abstract of a paper on this subject recently presented to the Royal Society, and which contains a detailed account of a series of observations on the effects of localised lesions of the brain of monkeys, made partly by Drs. Ferrier and Yeo, and partly by Dr. Ferrier alone.

The operations were performed under anæsthetics, and according to the rules and methods of antiseptic surgery.

The experiments are illustrated by photographs, showing the position and extent of the lesions and general condition of the brains, together with sun-prints and microphotographs of sections of the brain and spinal cord, showing the structures primarily involved or secondarily degenerated.

As the paper is mainly an anatomical description of the lesions delineated and record of the effects produced, it scarcely admits of condensation in the form of an abstract. The lesions are classified according to their position in the occipito-angular, temporal, Rolandic, frontal, and hippocampal regions respectively.

Among the more important results arrived at are the following:—

Lesions of the occipito-angular region (occipital lobes and angular gyri) cause affections of vision, without affections of the other sensory faculties or motor powers.

The only lesion which causes complete and permanent loss of vision in both eyes is total destruction of the occipital lobes and angular gyri on both sides.

Complete extirpation of both angular gyri causes for a time total blindness, succeeded by lasting visual defect in both eyes.

Unilateral destruction of the cortex of the angular gyrus causes temporary abolition or impairment of vision in the opposite eye—not of a hemiopic character.

Deep incisions may be made in both occipital lobes at the same

time, or the greater portion of one or both occipital lobes at the same time may be removed without any appreciable impairment of vision.

Destruction of the occipital lobe and angular gyrus on one side causes temporary amblyopia of the opposite eye and homonymous hemianopia of both eyes, towards the side opposite the lesion.

As in none of the cases recorded, either of partial unilateral or bilateral destruction of the occipito-angular region, were the amblyopic or hemianopic symptoms permanent, it is concluded that vision is possible with both eyes if only portions of the visual centres remain intact on both sides.

Destruction of the superior temporo-sphenoidal convolution on both sides causes complete and permanent loss of hearing, without other sensory or motor defect. Hearing is not impaired by lesions of any other part of the temporal lobe.

Destructive lesions of the Rolandic zone (convolutions bounding the fissure of Rolando) cause motor paralysis, without loss of sensation, limited (monoplegia) or general (hemiplegia), according to the position and extent of the lesion.

Lesions of this region are followed by descending degeneration of the pyramidal tracts of the spinal cord.

Lesions of the frontal region vary according as they affect the post-frontal (base of the frontal convolutions) or pre-frontal (anterior two-thirds of the frontal convolutions) region.

Lesions of the pre-frontal regions alone do not produce any discoverable physiological symptoms.

Lesions of the post-frontal regions cause temporary paralysis of the lateral movements of the head and eyes.

As the symptoms are only temporary so long as any portions of the frontal lobes remain intact, it is concluded that the post-frontal and pre-frontal regions have essentially the same physiological signification, and that portions only of the frontal lobes are sufficient for the exercise of their functions.

Lesions of the frontal lobes are followed by descending degeneration of the mesially situated tracts of the foot of the crus cerebri and corresponding fibres of the internal capsule, as seen in transverse sections. The ultimate destination of these tracts is uncertain. They cannot be traced in the anterior pyramids.

Lesions entirely destroying the hippocampal region (the hippocampus major and gyrus hippocampi) and neighbouring inferior temporo-sphenoidal region (without implication of the crus cerebri,

basal ganglia, or internal capsule) cause complete anæsthesia—cutaneous, mucous, muscular—of the opposite side of the body, without motor paralysis.

The degree of impairment of tactile sensibility in those cases where it was not entirely abolished, is in proportion to the amount of destruction of the hippocampal region.

Lesions of the cornu ammonis alone, or gyrus hippocampi alone, do not cause permanent impairment of tactile sensibility. The duration of the effects of total destruction of the hippocampal region has not been determined.

No impairment of tactile sensibility has been observed in connexion with any of the other cerebral lesions described.

A. DE WATTEVILLE.

Abstract of an Essay upon the Limitations in Time of Conscious Sensations. Read before the Royal Society, Edinburgh, December 1883. By JOHN B. HAYCRAFT, M.B., F.R.S. (Edin.) &c., Professor of Physiology at Mason and Queen's Colleges, Birmingham.—If any sensory surface be stimulated for a given period, a sensation will be produced, which will not exactly correspond with this stimulation in point of time. The sensation will be produced a certain interval after the first movement of application of the stimulus, and will remain a certain time after the withdrawal of the stimulus. It follows from this, that two stimuli follow the one another sufficiently rapidly will give rise to a single sensation. The correspondence in time between the stimulus and sensation varies in the case of the different senses.

In experimenting with tactile impressions rapidly following one another, it was found that if the stimuli are at first applied slowly (1 to 40 per second), they give rise each to a distinct and separate feeling. Stimulate more rapidly, and they are fused, and a feeling or sensation is produced which is termed "roughness." If the impacts follow one another faster than 1400 per second, the sense of "roughness" disappears and a sensation of single impact is produced. It is to be remarked that the knowledge of the cause of the roughness is gained by experience only, and could not be learned by the tactile sense alone. The same sensation called "tickling" is produced, whether an impact stimulates a number of sensory areas one after the other, as in drawing a feather over the skin, or if the impacts follow one another on the same sensory area; for impacts of from 50 to 1400 per second, if applied to the

same sensory area produce "roughness" if forcible enough, but when lightly applied, they produce "tickling."

Different areas of the skin vary in sensibility. In more sensitive parts localisation in space is more precise than in those parts less sensitive. The experiments of the author, however, show that in the least sensitive parts, *i.e.* skin of the back and chest, stimuli following one another rapidly are as easily distinguished in consciousness as when applied to the more sensitive parts, *i.e.* skin of lip and finger-tip. A study of thermal sensation shows that the correspondence in time between the stimulus and produced sensation varies with the thinness of the epidermis at that part. In no case is the correspondence very exact, an appreciable interval intervening between the application of a cold body and a sensation of cold. The difference in the exact limits of the feelings seen in the different sensations was then shown by the author to be due not to any difference in the centrally produced feelings, but to the differences seen in the translation of the external energy into nerve energy through the end organs. Thus a sensation of touch follows almost immediately the application of the stimulus, which directly affects the end organs; on the other hand, some time elapses before a cold body is felt; the withdrawal of heat from the end organs having to take place through a more or less thick layer of epidermis.

In the sensations of sound and sight, as well as in the tactile sensibility, when stimuli are repeated more and more rapidly, a period preceding complete fusion is reached when the sensation produced is disagreeable in its nature. In the case of sound, the irritation is produced by "beats;" the cause of "dissonance" in music is due to recurring stimuli not to be separated in consciousness, and yet not rapid enough to give rise to a simple sensation of sound. The flickering of a flame is also irritating in its nature, and tickling in the case of tactile sensibility may give rise to excruciating agony. We may compare, therefore, roughness or tickling—tactile sensations—to dissonance in music.

J. B. Haycraft.

Adamkiewicz on Cerebral Pressure (*Sitzungsber. der Akad. d. Wissensch. zu Wien*, 1883).—The author has had the opportunity of observing a tumour of the cord which occupied the greater portion of the left lateral column between the 5th and 7th cervical roots, and compressed the anterior horn to a marked degree. No symptoms had been noted during life, however, which gave a clue to such a lesion. This case formed the starting-point of investigations by

the author concerning cerebral pressure on the one hand, and the question of compressibility of nervous masses on the other. The first point only is discussed in the present paper. He concludes that a pathological increase in the pressure of the cerebro-spinal fluid (in other words in the "cerebral pressure") does not exist. This view is in complete opposition with the generally accepted theories on the influence of agents which diminish the intra-cranial space; but is supported by a series of experiments too considerable for us to adduce or criticise here.—The author describes the phenomena usually ascribed to cerebral pressure being due to cerebral irritation.

Sigmund Fuchs on the Histogenesis of the Human Cerebral Cortex. (*Ibid.* 1883).—The chief questions which the author has proposed to himself are: (1) At what period of the fœtal or extra-uterine life do medullated fibres (chiefly those which run parallel to the surface) make their first appearance in the cerebral cortex? (2) At what period are their number and calibre so far developed as to represent a system of fibres functionally equivalent with that in the adult?

He came to the following conclusions:—The new-born child possesses no trace of medullated fibres, neither in the central nor in the cortical portions. They are found in the former at the end of the first month only; in the cortex they appear at different times in the different layers. In the upper layer none are discovered before the fifth month; in the second layer, after the first year. The radiating bundles of the deeper layers appear in the second month; whilst the horizontal fibres of the third layer are found to be medullated in the eighth month.

During the seventh and eighth year the medullated fibres reach the disposition which they preserve throughout adult life.

The cortical medullated fibres, which in after-life are the thickest, are the first to make their appearance. We may assume that the chronological differences in the development of the medullated fibres correspond to their physiological interpretation. Pyramidal cells of the typical structure occur in the cortex at the time of birth. Deiter's connective tissue cells were clearly observed in the fifth month of child-life.

Zuckerkandl on the influence of the growth of Sutures and of the Cranial Shape in the direction of the Convolutions (*Medic. Jahrb.* 1883, Part IV.). Many observers have tried

to prove that there are certain relations between the shape of the skull and the direction of the convolutions; in the dolichocephalic brain they thought it to be more longitudinal, in the brachycephalic more transverse. If this be the case, the usual direction must be departed from whenever external circumstances (such as intra-uterine pressure, premature synosteosis of the sutures, &c.) alters the normal development of the skull. The author closely examined six brains from skulls deformed by synosteosis; he found the altered shape of the brain persists after removal, and that the convolutions present the alterations just described. He also examined the brain of a child whose head had been subjected to abnormal pressure "in utero," on account of the deficient amount of amniotic fluid, and observed similar phenomena.

We may go still further and show that under normal circumstances the sutures exert an influence upon the convolutions. The frontal gyri have not everywhere the same shape, breadth, thickness and direction. Quite in front they are usually narrow, thrown into numerous folds, and connected with one another by transverse pieces. Posteriorly, when they pass into the ascending frontal they are wide, thick, much straighter. This dissimilarity is easily explained by the topographical relations between the convolutions and the coronal suture. As long as the skull grows rapidly, the convolutions will lengthen longitudinally wherever the skull expands in that direction; this is the case for the posterior part of the frontal gyri, which are situated in the region of the coronal suture. Anteriorly and inferiorly, however, where no suture allows of such a ready expansion, the growing convolutions must become more concentrated, closely pressed together and thrown into folds. Similar considerations obtain with reference to other parts of the cerebral surface.

V. Rogner, On the Variations in the Cerebral Fissures in the Hare, the Sheep, and the Pig (*Zeitschr. für Wissensch. Zoologie, vol.* 39).—Numerous variations are to be observed in the disposition of the cerebral sulci of these animals, even on the almost smooth brain of the hare. The author reaches the important conclusion, that these variations are not to be considered as mere anomalies, but as transitional forms between the type of sulci in one kind of animals, and that observed in other kinds.

H. Obersteiner, M.D.